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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

PUBLIC HEALTH ADMINISTRATION IN WESTERN UNION COUNTRIES: A COMPARATIVE SYNTHESIS*

Neville M. Goodman, M.D., F.R.C.P., D.P.H.

On the 17th March, 1948, a Treaty of Economic, Social and Cultural Collaboration and Collective Self-Defence was signed at Brussels between the Governments of Belgium, France, Luxembourg, the Netherlands and the United Kingdom, to remain in force for 50 years.

Under that Treaty, a Permanent Commission with a small Secretariat was set up in London and Committees of Experts on various subjects were appointed, to further the objective of Article II of the Treaty, which was "To develop on corresponding lines the social and other related services of their countries."

One of these—the Public Health Committee—early decided that one of its major objects should be to provide mutual information and personal contacts through interchange visits of public health officials. "Development on corresponding lines" would be impossible without mutual knowledge of the position in the other countries.

As United Kingdom medical representative on the Public Health Committee, it was my good fortune at the end of last year, with an administrative colleague, to make the initial visits to the other four countries, to gain a general picture of their public health administration and prepare the way for other more specialised visitors. The information gained on these visits forms the main source for this lecture.

The general economic and social position of Western Europe, as a group, is that of a relatively densely populated and wealthy area, with, of course, a long history of industrial, social and cultural development and public health activity.

If the individual countries are compared, considerable differences both in wealth and in density of population are found. France has the double disadvantage, from the point of view of the organisation of health services, of a thinly distributed population and relatively low average real wages.

Turning to the general attitude of the five countries to administration, we can, I think, distinguish between the French and Belgian philosophy, which regards administration as a science, and the British, which considers it an art—or even a craft: with the Dutch, it seems, somewhere between the two. In other words, the Franco-Belgian approach is logical and sometimes—in British eyes—impracticable; the British approach is practical and nearly always illogical.

The British also tend to put greater emphasis on persons and the personal approach and contact, whereas the French feel that the most important point is that the project should be soundly and logically based and the various positions and functions properly defined. The expert is regarded with greater distrust in Britain than on the Continent, and perhaps the phrase "a sound man"—the highest encomium which the British administrator can receive, and which has no exact French equivalent—reflects this greater mistrust for the man "far gone in enthusiasm"—or special knowledge.

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* A Chadwick Trust Public Lecture, delivered on Oct. 24, 1950.

Moreover, the administrative systems, including public health administration, have been profoundly influenced by the historical development of the respective countries. Thus the Dutch tradition of individual and local self-reliance and effort has tended, as Dr. Banning showed so clearly in a recent Chadwick lecture,(1) towards a conservation of voluntary effort in public health and other fields. The French—and to a lesser extent the Belgian—systems owe much to the Napoleonic concepts of the " droit administratif " and of centralisation, while the British tradition of sanitary reform—essentially we feel a matter for local authorities—has fixed the broad lines of our public health system on a local basis; the story of the revolt against Chadwick's reforms is an example of this, and even the introduction of the National Health Service shows it; for example, the Minister first centralised the hospital services, and then immediately decentralised them as much as possible.

Considering the countries in turn, let us first deal with the special position of Luxembourg.

The *Grand Duchy of Luxembourg* is hardly comparable to the other four countries since—though a completely independent country—it is only the size of an average English county, having the area of Derbyshire and about half the population.

Public health is entirely centralised under the Ministry of Public Health in the capital. The Communes, which are the administrative divisions of the country, have no health functions. The Ministry of Health was created 20 years ago—but was only separated from the Ministry of Justice after the last war. The Minister himself performs some of the duties which elsewhere would be undertaken by the Permanent Secretary or Director-General of the Ministry and, since all seven Ministers combine several functions, he also is responsible for war damage—still an important problem in northern and eastern Luxembourg. Social assistance and physical education used to be in his province but were removed a few years ago.

There is a lay head of the non-medical and administrative activities of the Ministry—a woman—but public health is in the charge of a medical director (Médecin Directeur) Dr. L. Molitor. In fact, Dr. Molitor is—himself and alone—public health in Luxembourg. He has no medical assistants except for the Director of the Department of Health Laboratory, though he hopes to obtain three assistant medical officers shortly. He deals with epidemiology and controls the central staff of disinfectors and health visitors; with tuberculosis and venereal disease; with the State hospitals for tuberculosis, mental disease and maternity; and is concerned with an immense variety of other questions, including the external relations of the Grand Duchy in health matters, e.g. he represents Luxembourg on the World Health Organisation and the Western Union Public Health Committee.

To translate this into English experience, it would be as if the county medical officer of health for Derbyshire, with no assistant county medical officers and no medical officers of health in the county borough or county districts, carried out the duties of all of these officers and in addition represented the Grand Duchy of Derbyshire at international health conferences! Dr. Molitor's energy and abilities, however, go far to make up for its exiguous staffing—and he is a frequent visitor to this country and is familiar with our public health system, as well as with those of the other three Western Union countries. Thus, he would be in a position to pick out the best in the other four countries and apply it to Luxembourg—if he were given the means.

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(1) Banning, C. (1950). Co-ordination between the Public Health Service and private enterprise in Holland: its results. Lond. Chadwick Trust.

His tasks are lightened in some directions, e.g. by the coal and iron industries in the south-west having their own efficient medical service; by the general hospitals in the country being either municipal or voluntary (religious); and by the school medical service—such as it is—not being his responsibility. All doctors and nurses (but not practical nurses and midwives) are trained outside the country. There is a National Council to co-ordinate the maternity and child welfare work, which is largely run by voluntary organisations; medical and pharmaceutical practice is supervised by a Collège Médical. At present some 70 per cent. of the medical students train in France, 15 per cent. in Belgium, 10 per cent. in Switzerland and a few are beginning to go to Germany again.(2)

Leaving Luxembourg, let us first consider and compare the *Central Health Departments* of the other four countries, called the Ministry of Health in England and Wales (Department of Health for Scotland, Ministry of Health and Local Government in Northern Ireland); the Ministry of Public Health and Population in France; the Ministry of Public Health and the Family in Belgium; and the Ministry of Social Affairs in the Netherlands. All of these were created between the wars, that of the United Kingdom being the earliest and the most stable—the other three having suffered various changes of name and functions. Of course, certain central public health functions were carried out long before the creation of special Ministries, France for example, having had an epidemic control section in the Ministry of Commerce as early as the 18th Century, and Belgium an industrial hygiene service since 1810.

All four Ministries have additional functions besides health. In England and Wales, for example, housing and relations with local authorities, which latter are in the other countries matters for the Ministry of the Interior. In France, the Population section of the Ministry of Health and Population handles public assistance (not social security), demography and immigration. In Belgium, the Family section of the Ministry of Health and Family is also responsible for public assistance, housing, physical education and sport; while in the Netherlands, health forms only two out of eleven sections in the Ministry of Social Affairs, the others including employment, labour protection and relations, social insurance and public assistance.

The main differences in *structure and organisation* are as follows:—

Whereas all are in charge of a Minister, usually with a Deputy Minister or Parliamentary Secretary (the latter often charged with the oversight of particular subjects), in France and Belgium the Minister's Office (Cabinet du Ministre) has great power and its head, the Chef du Cabinet, is a political appointee, usually changing with the Minister: no similar arrangements exist in Great Britain or the Netherlands. There seems a tendency on the Continent for Ministers of Health to belong either to the extreme Right or the extreme Left of the Government in power.

Secondly, in the sections dealing with health, in all countries except the United Kingdom, doctors and other technical health staff are eligible for, and in fact hold, executive positions on a par with non-technical administrators. Thus in France, Belgium and the Netherlands, the permanent heads of each of the Ministries are doctors, as are most of the directors of the divisions dealing directly with health: where they are not, the deputy director is usually a doctor.

A special and important feature peculiar to France, which dates from pre-Revolutionary days and originates from a fundamental principle of French administration, the separation of "administration" from "control" or "inspection," is the corps of *General Inspectors*—l'Inspection Générale. There are nine General Inspectors in the French Ministry of Health, five doctors and four administrators, with equal status except that the senior acts as their head. They are appointed permanently by decree of the President of the Republic on the proposal of the Minister and have executive powers, reporting directly to the Minister, of whom they have been described as the "eyes and ears". Their duties have been described as:—first, to inspect all the services dependent on the Ministry; secondly, to go on special missions of enquiry, even outside the country, particularly to investigate "incidents" on behalf of the Minister, which might have political repercussions: (examples given were an enquiry into the poliomyelitis epidemic, and a mission to Réunion, recently created a French Department, to unify its health services with those of Metropolitan France) and, thirdly, to conduct collectively a special investigation each year into one subject of outstanding importance, e.g. in 1948 into the cost and organisation of public assistance; in 1949 into the care of abnormal children.

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(2) Molitor, L. (1948). Rapport sur la situation sanitaire au Grand-Duché de Luxembourg depuis la Libération. Luxembourg, March 1948.

In France there are five Divisions (Directions or Directions-Générales), viz. (1) General Administration, Staff and Budget; (2) Public Health and Hospitals; (3) Social Hygiene; (4) Pharmaceutical; (5) Population. These are sub-divided into various Offices (Bureaux). For example, in the Division of Public Health and Hospitals there are ten bureaux, the Public Health Sub-Division having six bureaux for (1) the practice of the medical and para-medical professions; (2) epidemiology; (3) water, sanitation, cleansing, and the Superior Council of Hygiene; (4) laboratories and spas; (5) quarantine and international relations; (6) nurses: and the Hospital Sub-Division having four, i.e. hospital construction; equipment; administration; administration and finance of special hospitals.

In Belgium, there are two all-purpose Divisions, i.e. legal and documentation, and administration, which serve all the four other divisions, comprising hygiene; social medicine; family, housing and assistance; physical education and sports.

In the Netherlands, the two departments directly concerning health are National Health and International Health, the former with thirteen bureaux.

The structure in England and Wales is somewhat different, partly because of the National Health Service, but chiefly because the medical and technical divisions are separate and advise the administration (or executive divisions) as necessary, and are not intermingled with them.

In France, Belgium and the Netherlands there is a clear-cut division between the Internal and External Services of the Ministry.

To detail the apportionment of functions and titles of the various health sections of the four Ministries is unnecessary, since the work to be done is essentially the same and the method of allocation is relatively unimportant and often changed.

We must next mention the *other Ministries and Departments having health functions*. In addition to the medical services of the Armed Services and the Colonies, in France and the United Kingdom there are the school medical services which come under the Ministry of Education, while in Belgium and the Netherlands they are still a local function. Industrial hygiene is the concern of the Ministries of Labour everywhere (though the Secretary-General of the Belgium Ministry of Health, Professor de Laet, is Professor of Industrial Hygiene in the University of Brussels). Health insurance in Belgium and France is also not under the Ministry of Health.

In this country the establishment of a system of statutory *Advisory Councils* to the Minister on health questions is comparatively recent and developed from the establishment of the National Health Service: we had previously relied much more on *ad hoc* committees. In the other three countries, however, such Advisory Councils are of great importance and considerable antiquity. Thus in France there are three such bodies in the main, the very distinguished scientific body, the Academy of Medicine, dating from 1820; the Higher Council of Public Health (Conseil Supérieur d'Hygiène Publique) dating from 1902; and the more recent Permanent Council of Social Hygiene. All three *may* be asked for advice by the Minister in their respective spheres and *must* be consulted by the Minister on certain subjects, for example health legislation. The Conseil Supérieur d'Hygiène Publique must approve the establishment of individual water undertakings, abattoirs, etc., and that of Social Hygiene, sanatoria, preventoria and maternity and child welfare institutions. They also have the right to initiate advice to the Minister on any health subject. In Belgium, the two Royal Academies of Medicine (Flemish and " National ") and the Conseil Supérieur d'Hygiène dating from 1849, have similar functions and there are no less than 24 other official advisory commissions covering nearly every activity in public health and social medicine. In the Netherlands, there is a Health Council and Central Health Committee with the same chairman and secretary, the former for scientific and the latter for administrative questions. At the time of my visit, these bodies in all three countries had been asked to advise on the harmful effects or otherwise of coca-cola.

The systems of control of the medical and para-medical professions vary much. In the United Kingdom, disciplinary and ethical matters—deontology—are controlled by an old-established statutory body, the General Medical Council, and, to some extent, in the case of specialists, by the three Royal Colleges, while the British Medical Association looks after the material interests of the profession. In France, the creation of a Guild of Doctors (L'Ordre des Médecins) was much discussed between the wars and was finally created in 1945, after Marshal Pétain had abolished the trade unions, including the medical syndicates. L'Ordre des Médecins is organised on departmental (i.e. county), regional and central levels. The Regional Councils have disciplinary powers ranging from a warning notice to removal from the Register, subject to appeal to the National Council. Hearings are private. There is a legal code of ethics—Code de Déontologie—instituted in 1947. The material interests of the profession are looked after by the revived medical trade unions, the Syndicats medicaux. Departmental syndicates are grouped into a National Confederation and dentists, pharmacists and midwives—but not nurses*—are similarly organised. In Belgium the system is similar to France, whereas in Holland there is a Medical Association and a separate statutory court for professional offences, with a varying scale of penalties, composed of doctors with a legal chairman.

Turning now to *local public health administration* outside the central health authority, we find a fundamental difference between the three Continental countries and ourselves. This can be summed up in the word " centralisation " and arises from the British conception of public health as basically and essentially a local function, the central authority only taking on public health duties as it were reluctantly and as a *pis aller*, whereas abroad the arm of the central government has always stretched all over the country acting through prefects or governors and hence it has seemed natural that—except in the older and larger municipalities—the medical officer of health should be a state employee and have a dichotomy of function, looking both to the central government and also to the local council and its chief officer. In the United Kingdom, the position was somewhat similar to the Continental during the last war, with the appointment of regional commissioners and their principal regional medical officers. In the Netherlands the situation is similar to the Franco-Belgian centralised system in this respect, although in other matters they depend so largely on voluntary organisations.

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* Nurses are included in the Hospital Officers Union.

The basic *public health administrative divisions* in France are the department corresponding to our county, and the commune which, if a town, may also be called a municipality. The arrondissement, a division of the department, has no public health functions. The commune as such also has practically no public health duties but, where the commune is a municipality of a certain size, it takes over the bulk of the public health work from the department. All municipalities with over 20,000 population (over 5,000 in the case of spas) must have a health office (Bureau d'Hygiène), with a part-time medical officer of health: with over 50,000 population he should be full-time. The part-time medical officers should have a special qualification in public health but this is not the case everywhere yet. The medical officer of health or director of health of a municipality is a municipal employee appointed by the mayor. The part-time ones have no security of tenure but the full-time officers are sometimes seconded state health inspectors.

The department is governed by the prefect and an elected council (Conseil Général) which votes the budget. Every department has now a full-time director of health, qualified in public health, as well as a non-medical director of population. Though both are officers of the Ministry of Health and Population, within the department they are subject to the prefect's authority. The director of health has one or more assistant medical officers, working either on a geographical or functional basis, usually the latter. The office staff may be state or departmental employees.

The *functions* of the director of hygiene of a department fall under the three heads of policy direction, executive control and advice. For the whole department—excluding municipalities with separate health bureaux—he is responsible both for the environmental health services, including infectious disease, and for much of the newer personal services—in other words broadly for the duties which used to be carried out by both the county and county district medical officers of health in the United Kingdom. He also has many responsibilities for the hospitals, including holding the competitions (concours) by which all hospital posts are filled. In his consultative capacity he advises the departmental health committee—a committee of the departmental council ; attends meetings of the hospital governors ; advises on school medicine and consults with the Order of Physicians on the admission of doctors to the local register.

Since many of the 89 departments of Metropolitan France are too small for comprehensive health services, particularly hospital services, there is a strong tendency at present to revert to the regional organisation instituted by Marshal Pétain, but abandoned except in the organisation of the Order of Physicians and the National Health Insurance (Caisses). The new hospital organisation is being planned on a regional basis.

The set-up in Paris—with its division into arrondissements, which may be compared with London's metropolitan boroughs—is peculiar and so excessively complicated that space does not permit description.

In *Belgium and Holland,* the organisation is similar, with provinces for departments and governors for prefects, and other minor variations. However, in Holland the provinces have supervisory rather than executive powers and the commune—in contrast to British local authorities—may do anything for the public good which is not either forbidden by Parliament or allocated to another authority. This gives great scope for local enterprise and experiment.

In Belgium, I had the privilege of spending a day with the health inspector of the Province of Liège and following his ordinary daily routine. As stated above, it closely resembled the routine of a county district medical officer of health in the United Kingdom—though the district was much larger—with certain added functions such as V.D. control, i.e. it dealt primarily with such matters as infectious diseases, vaccination and immunisation, bad housing, water supplies, sewerage and refuse disposal and the inspection of " classified establishments ", i.e. factories, abattoirs and, notably, hospitals and clinics. Separate inspectors, of equal status, dealt with the inspection of food, meat and drugs.

The provincial inspector of health had a medical deputy inspector on his staff, three health visitors and two social assistants and a number of disinfectors. A major difference of course between this country and all the others is the absence of the sanitary inspector. It was strange to find the health visitors dealing even with reported nuisances and bad housing.

Two other points struck me, that the provincial health inspector had just closed under his own personal authority, a slaughter house which had failed to carry out the necessary sanitary improvements ; and that almost the only health function retained by the commune was the school medical service, performed by part-time general practitioners and naturally of very varying development.

The health services in the City of Liège were similar, with some additions, such as an ambulance service, a premature infant service, and the inspection of food and milk. The medical officer of health (director of hygiene) was a paediatric specialist, part-time.

Finally I may mention that the statutory annual report of the medical officer of health does not appear to be of anything like the same importance in the other three countries as it is in England and Wales.

To sum up the local health organisation, it can be said that in all four countries the actual health work done is naturally very similar, the chief differences being organisational, perhaps the most striking being the difference in status of the medical officer of health, who in the United Kingdom is usually a whole-time, specialised officer of the local authority at all levels, but in the other countries is a local authority officer only in the larger towns, where he is often a part-time clinician, whereas in the rest of the country he is a civil servant.

Something must next be said about the *general medical care* systems in so far as they are relevant to our subject. These comprise personal or domiciliary medical care, and care in hospitals and institutions.

But for the establishment of the National Health Service in the United Kingdom, the organisation of both domiciliary and hospital medical care would have been very similar in all four countries. As it is, personal medical care in France, Belgium and the Netherlands depends on a combination of free enterprise, national health insurance and public assistance ; and hospital care on voluntary, local authority and public assistance hospitals, apart from a few special state institutions. The pre-National Health Service organisation in this country, therefore, gives a fairly accurate picture of the present organisation in the other three, though the services provided by their sickness insurance systems are considerably more extensive—including hospital and specialist care for instance—than those of our old National Health Insurance. One major difference is that in France and Belgium the family doctor is not paid by capitation fee but directly by the patient, who is reimbursed a large but varying percentage by the insurance funds. Another difference is that in the Netherlands, the State does not contribute directly to the insurance fund, though it makes up the annual deficit.

On the *hospital* side, apart from the United Kingdom, the chief difference between the other three countries is that the proportion of voluntary hospitals, including religious, is much lower in France than in Belgium or the Netherlands. In France, the great majority of the hospitals are municipal or communal, a few being departmental, voluntary or state—the last chiefly psychiatric and sanatoria. Paris, Lyons and Marseilles have special systems. There are about four beds for acute cases per thousand population, but the distribution varies between departments from 7 to 1.5 per 1,000. Hospital re-organisation is a major preoccupation in France at the moment and, under a new proposed law, a re-grouping is being undertaken into four classes: (1) regional teaching hospitals grouped round the twelve medical schools; (2) departmental hospital centres; (3) hospitals, (*a*) general, for about 50,000-75,000 population; (*b*) incomplete; (*c*) small, i.e. a few maternity and other emergency beds only; (4) homes, for the aged and infirm. Dr. Bridgman of the French Ministry of Health has described this in greater detail in a previous Chadwick lecture.(3) The re-organisation will not be compulsory, but since the State repays some 40 per cent. of the gross hospital expenses, it can bring this about gradually by financial pressure.

In Belgium up to 1900, the vast majority of hospitals were public, i.e. belonged to the public assistance committees. Since then nearly all new hospitals have been private, i.e. voluntary, religious or profit-making. At present there are 112 public and 276 private hospitals, not including sanatoria, mental or homes for the aged. This gives about 3.4 acute beds per 1,000 population. There are no specialised hospitals or children's hospitals. Great improvements have taken place recently, partly from the fact that, since 1933, public and private hospitals are " classified establishments " which the state inspectors of health have a right to inspect, and partly because in 1945 it was decided that the hospital costs of insured patients would not be paid unless the hospital was approved by the Ministry: since some 80 per cent. of the patients are insured, failure to reach the standards laid down by the Ministry would mean closing the hospital. In addition, a programme for providing 6,000 additional beds in public assistance hospitals over the next ten years is now under way.

In the Netherlands, some 80 per cent. of the hospitals are voluntary, chiefly religious, the remainder being municipal, a few state and one university. Some integration has been achieved through the recent creation of a joint board, with state advisers, of the two National Boards of the Protestant and Catholic Hospitals. Hospital revenue comes chiefly from the sickness insurance funds, the State giving a grant of 3/7ths to approved capital expenditures.

Time will not permit a comparison of special aspects of the public health services such as epidemiology, laboratories, maternity and child welfare, V.D., tuberculosis, and nursing, except to mention that maternity and child welfare and tuberculosis control, both preventive and curative, are in Belgium almost entirely in the hands of national voluntary organisations, very heavily subsidised but not controlled by the Ministry of Health. The large part in the preventive health services of the Netherlands played by the voluntary Coloured Cross Organisations has already been stressed by Dr. Banning.

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(3) Lancet, 1949. i, 194.

The basis of any public health service is the training of its officers and a word about the *training of doctors in public health* in the four countries is necessary.

The instruction of medical students—undergraduates—in public health is similar in all four, though I formed the impression that it was more thorough on the Continent than at least in some schools here, where the amount of time devoted to public health varies very greatly.

In postgraduate instruction, however, we have had a statutory course and diploma in public health controlled by the General Medical Council for 64 years, whereas it is only recently that any uniformity has been brought into the postgraduate courses and diploma in hygiene in the medical schools in the other countries. In the Netherlands, such postgraduate instruction is only beginning this year.

On the other hand, the creation of a French National School of Public Health in 1947 in Paris and the striking fall in the number of D.P.H. students in this country since 1948 suggest that we may be in danger of losing our long-standing lead in public health training: indeed unless the position here changes, it may be impossible to fill the vacancies for public health medical officers, which is already causing difficulty in the central services.

In conclusion, may I recapitulate the more salient points in the four countries as they struck me.

In France, within the Ministry of Health, the special position and importance of the Minister's Office and the Chef du Cabinet ; and the corps of general inspectors and their duties ; the first being also found in Belgium. Outside the Ministry itself, the special position of the consultative bodies, the Order of Physicians and the National School of Public Health—the first two being also in the Belgian system.

The Belgian organisation is fairly close to that of France, a special point being that maternity and child welfare and tuberculosis are entrusted to voluntary organisations.

In the Netherlands, in one sense the set up is in general more akin to this country, except for the great part in preventive medicine played by the voluntary Cross Organisations. On the other hand, a National Health Service such as ours is perhaps an even more remote possibility in Holland than in France or Belgium.

All three countries differ from our own in the appointment of central government officers in charge of local areas ; in the exercise by both technical and administrative officers of executive powers ; in the part played by sickness insurance and public assistance in medical and hospital care.

Each of the Western Union countries has good and less good points in the structure of its public health organisation and each has certainly something to learn from the others. We are too apt, perhaps, to think of something as strange and peculiar because we do not do it ourselves, whereas enquiry will show that it is we who are peculiar or in a minority. We would all agree I suppose, for example, that our National Health Service is peculiar, because it is new and we have not yet grown up with it. But we also may feel it is peculiar of the others not to have sanitary inspectors, though we are almost alone in the world in having them: or we may think it peculiar to give executive powers to doctors in central health departments ; though Ethiopia is, I believe, the only other country in the world, including the Dominions, which follows our example. I suggest that the " convergent development " aimed at by the Brussels Treaty is, in public health, an objective attainable only if we begin by learning something of the organisation in the other Western Union countries.

SURVEY OF SICKNESS—JUNE QUARTER, 1950

(Issued from the General Register Office, Somerset House, W.C.2)

The Registrar General's Quarterly Return No. 407 (in Tables A to H) gives detailed results of the Social Survey's interviews of random samples of the adult population carried out during the months May to August, 1950, in which the experience of April to June was recorded. The report given here presents some rates derived from these tables without standardization or correction for the number of days in the month and compares them with those for preceding months. More detailed studies will appear in the publications of the General Register Office.

The Monthly Health Index—the proportion of persons, per 100 interviewed, who suffered no illness or injury so far as they remembered during the stated month—is given in Table A, separately for men and women at ages 16-64 and ages 65 and over. At ages 16-64 the indices for the three months from April to June, 1950, were almost the same as during the corresponding months of 1949 but were lower (worse) than during the same period of 1948. At ages 65 and over the indices for the June quarter, 1950, were rather lower than during the corresponding period of 1949 and as in the case of younger persons, considerably lower than in 1948. Only 21 per cent. of the elderly men interviewed reported having had no illness during the month of June, 1950, compared with 30 per cent. during June, 1948.

Table B gives monthly morbidity rates (the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only), days of incapacity and numbers of medical consultations, distinguishing persons with an illness commencing in the month from those with an illness continued from the previous month. Including persons with injury, average rates for the June quarter of 1948, 1949 and 1950 compare thus:—

	Ages 16–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
Apr.-June, 1948 ...	61·3	73	35	79·0	133	58
Apr.-June, 1949 ...	65·8	79	39	83·1	159	65
Apr.-June, 1950 ...	65·1	81	42	85·0	137	76
Apr.-June 1950 per cent. of Apr.-June						
1948	106	111	120	108	103	131
1949	99	103	108	102	86	117

At ages 16-64 there was a slight increase in numbers of days of incapacity and of medical consultations in April to June, 1950, compared with 1949 but no increase in the proportion of persons ill. At ages 65 and over, although sickness and medical consultation rates increased, there was a substantial reduction in numbers of days of incapacity. During the month of June, 1950, elderly persons reported only 94 days of incapacity per 100 interviewed compared with 142 days of incapacity in June, 1949, and 108 days in June, 1948.

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during the month from those who developed an illness of a minor or ill-defined nature. Average monthly rates during the June quarters of 1948, 1949 and 1950 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	16–64		65 and over		16–64		65 and over	
	M	F	M	F	M	F	M	F
Apr.-June, 1948	3·5	3·8	3·6	5·5	26·7	34·1	26·5	29·9
Apr.-June, 1949	3·2	4·0	4·0	5·5	28·6	38·1	28·0	31·2
Apr.-June, 1950	4·4	4·9	4·9	7·6	30·8	39·2	30·5	35·7

At both ages each of the rates was rather higher in the June quarter, 1950, than in the June quarters of 1948 and 1949, and the rates in respect of minor and ill-defined illnesses increased each year.

During the course of the three months April to June, 1950, the rates of more serious illnesses declined. The proportion of women with minor illnesses, however, increased in May but declined again in June.

Table D gives average monthly numbers of new illnesses from selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age and the numbers of days of incapacity arising from each cause (including both new and continued illnesses). The incidence of colds and influenza during the June quarter, 1950, was much the same as in the corresponding period of the two previous years. The number of days of incapacity that they caused during the quarter, however, was a little greater than in 1949 and much greater than in 1948. Illnesses from other respiratory diseases and rheumatic complaints tended to be a little more frequent than in either of the two previous June quarters, but the amount of incapacity attributed to rheumatism was not as much as in the June quarter of 1949, especially among elderly persons.

Days of incapacity from all causes, shown in the bottom right-hand corner of Table D were only slightly more frequent at ages 16-64 during April-June, 1950, than in the two previous June quarters. The amount of incapacity reported at ages 65 and over was less than in April-June, 1949, and little different from April-June, 1948.

A.—*Monthly Health Index : The proportion of persons, per hundred persons interviewed, for each sex and age who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 16–64						Ages 65 and over					
	M			F			M			F		
	1948	1949	1950	1948	1949	1950	1948	1949	1950	1948	1949	1950
January	40	36	34*	32	26	25*	21	20	21*	13	12	12*
February ...	40	34	34	31	24	27	19	18	20	15	9	11
March	42	34	38	31	24	27	23	16	20	16	12	11
April	45	39	38	34	27	29	25	19	18	15	12	11
May	45	40	41	33	29	29	27	23	21	16	15	12
June	44	43	43	34	31	31	30	24	21	19	14	12
July	45	44		33	32		26	26		16	14	
August	47	45		34	32		25	26		15	16	
September ...	43	45		31	32		23	25		16	15	
October	39	40		28	27		24	24		12	14	
November ...	36	36		27	27		22	23		12	13	
December ...	35	37*		25	28*		17	27*		12	14*	

(Based on combined results of interviews in two following months.)

* Experience based on one month's interviews corrected by factors based on previous experiences.

B.—*Monthly Morbidity Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 16–64				Ages 65 and over			
	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month
1948								
January ...	39·8	24·6	95	38	39·2	43·6	224	64
February ...	39·9	24·9	105	40	38·9	43·9	239	74
March ...	37·4	25·8	87	41	36·4	44·4	199	67
April	34·3	26·4	73	35	35·2	45·5	152	67
May	34·7	26·0	75	35	31·7	47·8	139	61
June	34·3	27·1	71	37	32·5	44·3	108	48
July	34·9	25·9	74	36	33·8	45·3	133	67
August ...	34·0	25·6	69	33	32·7	48·0	163	56
September ...	37·6	25·2	75	35	35·4	45·6	155	48
October ...	41·6	25·0	101	42	40·1	42·5	193	60
November ...	46·0	22·3	105	43	46·2	37·0	285	74
December ...	47·0	23·0	103	42	44·4	41·0	334	76
1949								
January ...	44·5	24·5	110	46	44·2	39·7	217	72
February ...	46·9	24·2	131	50	44·7	41·3	207	71
March ...	45·9	25·2	128	46	43·7	42·1	211	83
April	39·7	27·0	84	40	38·5	46·5	175	74
May	38·2	27·1	75	40	33·1	48·1	163	61
June	34·5	28·1	77	38	32·6	47·8	142	60
July	33·4	28·4	78	41	33·1	46·6	118	63
August ...	32·5	28·4	74	38	32·7	46·9	71	54
September ...	35·9	25·2	74	39	35·9	44·6	96	60
October ...	44·1	22·5	93	42	40·6	40·9	212	59
November ...	47·1	21·0	102	41	42·8	39·4	293	73
December* ...	51·0	20·1	100	41	46·3	36·5	323	65
1950								
January* ...	43·9	22·5	113	42	42·2	39·7	212	68
February ...	46·8	22·3	117	50	43·9	40·3	211	77
March ...	44·7	22·2	107	49	42·2	43·3	221	87
April	41·4	24·0	86	41	42·6	43·5	183	85
May	41·0	23·5	79	42	40·5	43·9	133	80
June	37·4	25·3	78	42	36·9	47·0	94	64
July								
August ...								
September ...								
October ...								
November ...								
December ...								

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury, see Registrar General's Quarterly Returns, Table E.)

* Uncorrected rates based on one month's experience.

C.—*Rates of Morbidity per* 100 *Males and Females for illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
1948								
January	3·9	4·9	5·8	9·6	31·6	38·2	27·8	34·0
February	3·2	4·9	5·8	8·0	31·5	39·2	33·1	30·4
March	4·2	4·1	5·4	7·0	28·1	37·3	31·5	29·0
April	3·4	4·0	4·0	6·5	26·4	34·0	28·7	30·5
May	3·5	3·6	2·5	4·1	27·0	34·7	25·8	30·0
June	3·7	3·8	4·4	5·8	26·7	33·6	24·9	29·1
July	3·2	4·0	3·8	6·3	26·3	35·3	25·5	31·0
August	4·1	4·1	1·7	7·3	24·3	34·6	27·0	28·1
September	4·1	4·8	2·9	6·2	28·9	36·5	31·6	29·8
October	4·1	5·1	3·6	8·1	32·3	41·0	32·5	35·1
November	5·1	5·0	7·1	13·1	37·8	43·7	35·1	36·2
December	4·7	6·0	5·2	10·2	38·1	44·7	35·9	36·8
1949								
January	5·2	5·8	7·6	9·6	35·2	42·2	31·9	38·1
February	6·5	7·9	6·8	10·9	37·0	41·8	31·6	38·5
March	5·9	7·7	7·9	11·7	35·3	42·0	34·3	33·0
April	3·5	4·7	6·2	6·4	30·0	40·0	30·3	33·4
May	3·5	3·8	2·9	3·9	29·4	38·8	28·7	30·3
June	2·6	3·6	3·0	6·3	26·3	35·5	24·9	29·9
July	2·7	3·9	3·7	6·9	25·6	33·7	26·2	28·5
August	2·7	3·7	4·4	4·0	24·9	32·9	27·8	29·1
September	3·1	4·2	2·7	4·8	28·0	35·7	31·5	32·3
October	3·7	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November	4·9	5·3	8·5	9·7	38·3	45·1	32·7	34·1
December*	4·5	6·6	5·4	9·7	42·3	47·9	32·4	42·2
1950								
January*	3·7	6·0	9·6	8·0	35·4	42·0	28·0	37·2
February	5·9	8·1	9·2	9·2	36·1	42·8	31·4	37·0
March	6·1	8·1	8·4	11·3	32·4	41·7	29·4	33·8
April	5·3	5·6	5·5	10·6	32·9	38·6	33·7	34·4
May	4·7	4·9	5·5	6·6	31·5	40·3	29·8	37·8
June	3·1	4·2	3·6	5·7	27·9	38·6	27·9	34·9
July								
August								
September								
October								
November								
December								

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. " Ill-defined " excludes all symptomatic illness which caused incapacity for work, such cases being classed to the appropriate higher category. "Illnesses starting in each month" include new and recurrent illnesses.

* Uncorrected rates based on one month's experience.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity.*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1948								
January–March	14·8	16·1	11·5	11·4	12·0	15·2	12·9	14·1
April–June	9·1	8·6	5·5	7·5	4·7	6·0	1·6	8·7
July–September	9·8	9·3	6·1	6·7	3·3	5·3	1·9	3·4
October–December ...	20·0	20·3	12·2	13·9	12·9	16·6	9·3	23·9
1949								
January–March	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September								
October–December ...								
	Other respiratory disease							
1948								
January–March	2·7	3·1	4·4	5·4	8·5	6·4	24·3	27·4
April–June	2·8	2·6	2·6	3·3	7·5	4·2	8·0	7·5
July–September	1·8	2·7	2·7	2·6	3·3	2·4	10·1	9·6
October–December ...	3·6	3·3	4·8	4·9	9·5	7·3	24·2	22·0
1949								
January–March	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September								
October–December ...								
	Rheumatism, all forms							
1948								
January–March	4·4	6·1	7·0	8·5	9·1	7·4	19·6	12·9
April–June	4·1	5·9	8·2	7·3	3·6	2·7	4·7	16·5
July–September	4·3	6·6	8·1	7·1	4·9	4·4	9·2	5·9
October–December ...	5·5	7·8	9·9	10·7	5·1	4·6	3·7	20·9
1949								
January–March	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	8·2	22·2
July–September								
October–December ...								

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity*—(*continued*)

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Disorders of Heart and Arteries							
1948								
January–March ...	0·3	0·9	1·5	1·9	3·3	2·7	0·9	18·1
April–June	0·3	0·6	1·7	1·6	2·0	2·8	9·8	11·1
July–September ...	0·2	0·5	1·9	2·1	1·8	2·0	17·5	16·6
October–December ...	0·4	1·0	1·2	1·2	1·2	2·2	19·5	11·5
1949								
January–March ...	0·4	0·7	0·6	1·1	5·2	3·7	10·7	6·4
April–June	0·1	0·4	0·5	1·0	3·4	2·6	10·4	5·2
July–September ...	0·2	0·4	1·1	1·3	1·6	3·5	12·4	7·0
October–December ...	0·2	0·8	0·6	1·5	1·7	3·8	9·5	15·5
1950								
January–March ...	0·2	0·6	1·3	1·3	3·7	4·2	12·5	14·6
April–June	0·3	0·7	0·5	1·5	4·8	2·9	13·9	19·1
July–September ...								
October–December ...								
	Skin complaints				All illnesses and injuries			
1948								
January–March ...	1·5	1·1	2·0	0·6	110	85	203	235
April–June	1·8	1·2	0·7	0·9	85	63	128	136
July–September ...	2·1	1·2	0·5	1·2	78	68	102	184
October–December ...	1·7	1·2	1·6	1·0	100	105	183	344
1949								
January–March ...	1·3	1·4	0·4	0·9	119	127	200	220
April–June	1·7	1·4	0·8	0·9	84	74	151	165
July–September ...	1·6	1·0	1·0	0·7	81	71	89	99
October–December ...	1·6	1·3	1·1	0·5	95	98	225	302
1950								
January–March ...	1·6	1·5	1·4	1·0	111	114	196	231
April–June	1·9	1·4	0·8	0·8	88	75	143	133
July–September ...								
October–December ...								

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e., new, recurrent or continued from the previous month).

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, NOVEMBER, 1950

Issued from the General Register Office, Somerset House, W.C.2.

	Nov. 4	Nov. 11	Nov. 18	Nov. 25	Average weekly figures for Nov., 1949
Scarlet Fever	1,112	1,206	1,387	1,483	2,403
Whooping Cough	3,098	3,725	4,152	4,335	976
Diphtheria	49	55	76	61	97
Measles, excluding Rubella	7,821	8,857	10,139	11,357	1,228
Acute Pneumonia	433	465	558	490	583
Meningococcal Infection	32	26	33	37	23
Acute Poliomyelitis (Paralytic) ...	149	161	111	111	252
,, ,, (Non-paralytic)	57	64	39	19	15
Ophthalmia Neonatorum	35	26	38	45	31
Puerperal Pyrexia and Puerperal Sepsis	59	82	67	48	89
Dysentery	260	411	474	626	109
Paratyphoid	5	3	1	7	8
Typhoid	—	3	5	3	15
Smallpox	—	—	—	—	—

No cases of Cholera, Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

THE BACTERIOLOGICAL CONTROL OF DRINKING WATER

Memorandum No. 3 *of the Public Health Laboratory Service. Prepared by the P.H.L.S. Water Sub-Committee* as a synopsis and simplification of the Ministry of Health Report No.* 71 (1939).

This memorandum is not intended to replace Report No. 71, which remains the authoritative document on the subject, but it is designed to assist sampling and health officers who may have found the full report rather detailed. It is hoped that it will help them in making the best use of the bacteriological laboratory and in interpreting the results submitted.

Aim of Routine Bacteriological Examination

The greatest danger associated with drinking water is the possibility of its recent contamination by sewage or by human excrement. Most waters, in the natural course of events, are subject at some stage to sewage or manurial pollution. If the pollution is of human origin and of sufficiently recent occurrence, and if among the contributors there are cases or carriers of such infectious diseases as enteric (typhoid or paratyphoid) fever or dysentery, the water will almost certainly contain the living germs of these diseases ; the drinking of such water may result in fresh cases of the disease. When, by some mishap or breakdown in the normal precautions, these conditions arise in a public water supply, the consequence may be an explosive and widespread outbreak, such as the Croydon outbreak of typhoid fever in 1937. It may be noted that sporadic cases of enteric fever occur from time to time among bathers in sewage-polluted rivers and swimming pools (Reece, 1908 ; Manheimer, 1914 ; Harold, 1934 ; Martin, 1947).

Frank cases of disease are amenable to precautionary measures, but unrecognized carriers of disease are sufficiently numerous to keep the sewage effluents of populous areas regularly contaminated with pathogenic organisms. Though modern bacteriological methods have made it possible to detect these germs in sewage (Moore, 1948, 1950), it is impracticable to attempt to isolate them as a routine procedure from samples of drinking water. When present in fæces or sewage, however, the germs are almost always greatly outnumbered by the normal excremental organisms, such as *Bact. coli ;* in massed fæces or sewage the ratio of coliform to pathogenic organisms is probably never less than 1,000 : 1. These harmless organisms, moreover, survive rather longer in water, are somewhat more resistant to chlorination, and are easily detected.

The routine bacteriological examination of water, therefore, aims at the detection of fæcal pollution and consists chiefly in the search for *Bact. coli.* If this organism is not found in the water, it can with reasonable safety be inferred that disease-producing organisms are also absent. If the water contains large numbers of *Bact. coli,* dangerous germs may also be present and the existence of recent fæcal pollution is proved.

Apart from the dangers associated with the presence of *Bact. coli* in drinking water, the presence of any coliform organisms in the water used on dairy farms is undesirable, as they may reduce the keeping quality of milk through contamination of the utensils.

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* The P.H.L.S. Water Sub-Committee is composed of the following members of the P.H.L.S. Staff: R. D. Gray, M.D., D.P.H., (Chairman), W. H. H. Jebb, M.A., M.D., J. H. McCoy, M.B., B.Ch., D.P.H., J. Morrison Ritchie, M.A., M.B., Ch.B., D.P.H., A. J. Kingsley Smith, B.M., B.Ch., and Joan M. Watkinson, B.Sc.; together with E. Windle Taylor, M.A., M.D., D.P.H., Deputy Director of Water Examination, Metropolitan Water Board, London.

The Coliform Examination

Definition of coliform bacteria: By the term "coliform" is meant any organism which morphologically resembles *Bact. coli* and which by the usual cultural processes is liable to be confused with it. In practice this will comprise organisms of the *Bact. coli, Bact. ærogenes, Bact. cloacæ,* Intermediate and Irregular groups, and to a much less extent the Paracolon group; or, broadly speaking, those organisms that produce acid and gas in MacConkey broth within 48 hours at 37° C.

The search for *Bact. coli* commences with the presumptive coliform examination, which is conducted on a quantitative basis and reveals the presence of coliform bacteria of all types. Varying quantities of the sample (for drinking water the quantities are usually one of 50 ml. and five each of 10 ml. and 1 ml.) are inoculated into MacConkey broth and the tubes, after incubation at 37° C., are examined for the production of acid and gas. From the number of tubes showing this result the probable number of coliform bacteria present in 100 ml. of the sample is calculated, *i.e.* the presumptive coliform count. The positive tubes are then subcultured and the fresh tubes are incubated at 44° C. The production of gas at this temperature is practically specific for *Bact. coli* Type I (fæcal coli), and from the number of positive tubes is calculated the probable number of fæcal coli present in 100 ml. of the sample.

This is the basis of the modern routine examination, and, if sampling is frequent enough, it is all that is ordinarily required. Ideally, of course, all waters intended for drinking should show no coliform bacteria in 100 ml., but the coliform test is so sensitive that such a result represents a high standard of bacterial purity. Many natural waters do, and all chlorinated supplies should, give this result, but the universal adoption of this standard would debar many small supplies which have for years been consumed with impunity and for which no adequate alternative is available. In general, however, the presence of more than minimal numbers of fæcal coli indicates potential danger.

The Ministry of Health Report (1939), which deals at length with the whole subject, has suggested the following classification for piped water supplies, based on the presumptive coliform results:

Piped Supplies

		Presumptive Coliform Count per 100 ml.
Class 1.	Highly satisfactory	less than 1
Class 2.	Satisfactory	1–2
Class 3.	Suspicious	3–10
Class 4.	Unsatisfactory	greater than 10

Throughout the year 50 per cent. of samples should fall into Class 1; 80 per cent. should not fall below Class 2; and the remainder should not fall below Class 3.

In chlorinated piped supplies the water ought to fall into Class 1.

The Report recognizes that these criteria cannot be applied in rural districts where piped water is as yet unobtainable. "In such instances everything possible should be done to prevent the access of pollution to the water. By relatively simple measures, such as the removal of obvious sources of contamination from the catchment area, and by attention to the coping, brick lining, and covering of the well, it should usually be possible to reduce the coliform count for a shallow well water to a level between 10 and 25 per 100 ml."

Limitations of the Coliform Test

Although by far the most satisfactory test for faecal pollution, the coliform examination has certain limitations.

(1) Though *Bact. coli* Type I (faecal coli) is an undoubted faecal organism, occurring to the extent of, say, 100,000,000 per gram of human faeces (Houston, 1902), and has a short survival time outside the body, it is just as abundant in the excrement of animals and birds. The coliform test, therefore, readily detects animal pollution, which is usually less dangerous, and offers no means of distinguishing this from human contamination. It is never safe, however, to dismiss pollution which appears to be entirely of animal origin. In the first place, it is impossible to be certain that human pollution has not also occurred. Moreover, disease-producing organisms have been found in the faeces of domestic animals and birds (Cruickshank and Smith, 1949 ; Adams, 1928) and it is conceivable that water so polluted might give rise to disease.

(2) Although faecal coli form at least 90 per cent. of the total coliform organisms present in massed faeces or sewage (E. W. Taylor, 1949), the remaining 10 per cent. is composed of other types (intermediate, aerogenes, cloacae and irregulars) which are not so exclusively associated with faeces. These coliform types can survive longer outside the body, tend to be more resistant to chlorination, and in favourable circumstances may even multiply in such sites as filter beds or the jute packing of pumps and taps. They can often be found unassociated with faecal coli in soil and on vegetation, and there is some doubt whether they naturally inhabit these sites or whether their presence is due to their greater powers of survival. Their significance in water is thus somewhat obscure. If they are natural soil inhabitants which gain access to the bowel adventitiously with the food, their occurrence in water without faecal coli might indicate nothing more serious than the presence of soil washings. If, on the other hand, they are primarily faecal organisms whose wider distribution in nature results from their greater resistance, their presence in water which shows no faecal coli would indicate more remote faecal pollution from which faecal coli had already disappeared. Whatever their primary habitat, these organisms often occur in faeces in numbers which, though relatively much inferior to those of *Bact. coli,* may nevertheless be considerable (C. B. Taylor, 1942). When they are found in water, therefore, they must for safety be regarded as possible faecal indicators. It is for this reason somewhat unfortunate that they have acquired the term " non-faecal " to distinguish them from the more certainly " faecal " coli. There need be no confusion, however, in the continued use of these terms if it is clearly understood that, whereas faecal coli indicates recent faecal contamination, " non-faecal " coli without faecal coli indicates the possibility of less recent faecal contamination which is probably, for the moment at least, not dangerous.

Other Bacteriological Investigations

In some waters it may be advantageous to test for the presence of other excremental organisms, such as *Cl. welchii* or faecal streptococci ; but the modern tendency is to rely more and more upon the *Bact. coli* examination as being the most delicate and specific test of significant faecal pollution and to prefer repeated simple examinations of this kind to less frequent elaborate investigations. For the same reason, estimations of the general bacterial content by the plate-count technique are no longer considered to add sufficient information in ordinary routine control to warrant the extra trouble required.

Sampling

If the bacteriological findings are to be of any value it is imperative that the samples be properly collected. Scrupulous care is necessary to ensure that the sample is representative of the supply and to avoid the introduction of coliform and other bacteria from sources unconnected with the water under test. By imperfect sampling technique extraneous coliform organisms might get into the sample (*a*) from the sampler's hands, (*b*) from the interior of an improperly sterilized tap, or (*c*) from an open roof cistern—to give only three examples. The correct sampling procedure is fully described in an appendix of the Ministry Report, but it may be convenient to summarize the chief points as follows:—

(1) Use only the sterile bottles supplied by the laboratory.

(2) Keep the sampling bottle unopened till the moment of sampling.

(3) During sampling, do not let the stopper or the neck of the bottle touch anything. Hold the bottle near the bottom.

(4) Fill the bottle full, *without rinsing,* and immediately replace the stopper.

(5) Avoid leaking taps, which it is impossible to sterilize.

(6) Choose a tap which is directly connected with the main, and not one served from a roof cistern ; cisterns are sometimes open to contamination and should not be used for judging the quality of the main supply.

(7) Thoroughly sterilize the whole tap with the flame from a blow-lamp or cotton-wool plug saturated with methylated spirit.

(8) At the time of sampling, distinctively mark both the bottle and the application form.

(9) Transmit samples to the laboratory with the minimum possible delay. Samples must reach the laboratory in time for them to be dealt with on the day of collection. Any delay in commencing the examination may affect the coliform count.

Samples of chlorinated water for bacteriological examination should be collected in a sterile bottle containing a crystal of thiosulphate, which at once neutralizes any remaining free chlorine ; this is particularly important with swimming-bath waters.

Topographical Investigation

In the Ministry Report great stress is laid on the need for adequate topographical information: " No bacteriological examination, however careful, can take the place of a complete knowledge of the conditions at the sources of supply and throughout the distribution system." A topographical investigation should always be carried out, and possible sources of pollution should not be ignored, even though the results of the bacteriological examination are satisfactory. The technical limitations of laboratory analysis have already been noted, and sampling can provide only limited information. The examination of any sample can indicate no more than the conditions prevailing at the moment of sampling. A satisfactory result cannot guarantee that the observed conditions will persist in the future. On the other hand, it is manifestly superfluous to sample a well which by direct observation is liable to excremental pollution, until this fault has been remedied.

Every supply should be regularly inspected from source to distribution, and sampling should be repeated under varying climatic conditions, especially after heavy rainfall. Samples sent to the laboratory should be accompanied by full details of the topographical circumstances. This information is required by the bacteriologist for consideration in conjunction with his laboratory findings ; yet the bacteriologist is still quite often asked to assess the quality of a potential water supply on the basis of a single sample and the scantiest of information.

Sampling has its greatest value when regularly repeated. It may then confirm the purity of the water, or draw attention to the existence of hitherto unsuspected pollution. In the latter event an immediate investigation should be made to ascertain the cause of the deterioration. In difficult cases a personal inspection by the bacteriologist himself may be of help, but it is usually sufficient if there is close liaison between the sampler and the laboratory.

Chemical Analysis

The chief advantage accruing from bacteriological examination is the detection of recent faecal pollution. Chemical analysis, though lacking the delicacy of bacteriology in this respect, has a wide range of uses in the investigation of water supplies. It may reveal past pollution too remote for detection by bacteriological methods and can thus provide additional information towards the hygienic assessment of new supplies.

The services of a competent chemist are indispensable too, particularly with new supplies, for eliminating other possible dangers, such as lead solvency or the presence of poisonous metals, for determining the hardness and suitability of the water for domestic and industrial purposes, and for estimating, where necessary, the dose of chlorine to be used for purification.

Once a water supply is known to be chemically satisfactory and stable, further analyses (unless these are required for supervising treatment processes) need be made only at relatively infrequent intervals, because these, unlike the bacteriological tests, are not necessary for the day-to-day assessment of bacterial purity and safety.

The estimation of residual chlorine is also a chemical test. This, however, is a simple examination and is best conducted by the sampler at the time of sampling. Alternatively, arrangements can usually be made for this test to be undertaken on receipt of the sample at the bacteriological laboratory ; a suitable sampling bottle for this purpose will be supplied by the laboratory.

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TRACING A TYPHOID CARRIER BY SEWAGE EXAMINATION

N. C. Lendon, M.A., M.B., D.P.H., Medical Officer of Health, Havant and Waterloo Urban District, and R. D. Mackenzie, M.B., F.R.C.P.Ed., Dip. Bact., Director, Public Health Laboratory, Winchester.

During the spring of 1944 two boys were admitted to the Infectious Diseases Hospital, Portsmouth, suffering from typhoid fever (Vi-phage Type A). The evidence then available suggested that the infection had been contracted by drinking water from the Wallington River somewhere in the region of Pigeon House Lane Ford (Droxford R.D.). Bacteriological examination of the water provided ample evidence of recent heavy faecal pollution, although the attempts which were made then to isolate *Salmonella typhi* were unsuccessful. That the water should be contaminated was only to be expected, as the surrounding country was in military occupation and several large camps were situated close to the stream. In view of the difficulty of dealing with a transient service population and the imminent approach of D day, it was decided that the investigation could not usefully be pursued meantime.

However, the occurrence four years later, in the summer of 1948, of three further cases of typhoid fever (Vi-phage Type E1), among children who had independently visited the river and drunk its water, once again directed attention to the matter. Samples of river water were therefore collected from the places they had visited, and from three of these, Pigeon House Lane Ford, Comphouse Bridge and Mill Lane Bridge, *Salmonella typhi* Vi-phage Type E1 was isolated.

The Wallington River

> "*This is a secluded leafy country, with purling waters, and old brick bridges.*"—Local paper.

This river, which has several branches, flows in a westerly direction entering the sea at Fareham Creek. The southern branch, in which we were interested, rises at Crookhorn Farm just to the east of Purbrook (Havant and Waterloo U.D.) and, skirting this rapidly growing residential district in close proximity to a number of houses, passes under the London—Portsmouth Road at Purbrook Arches to enter Droxford Rural District. From then on it runs through pleasant country of fields and trees along the edge of the Forest of Bere until it enters Southwick Lake. Several small by-roads cross this picturesque stretch by fords and bridges, and in the neighbourhood of these picnic parties and children tend to collect; it was from water taken at several of these crossings that *Salmonella typhi* had been isolated. When the place was visited in June, 1948, the water level was low, and the banks which were freely exposed gave ample evidence of gross faecal pollution. It was therefore decided that a survey of the upper reaches of the stream should be made in order to try and locate the source of pollution, and if possible also the origin of the typhoid infection. This task was now considerably simplified since military occupation had ceased, and all camps had been removed.

The Upper Reaches

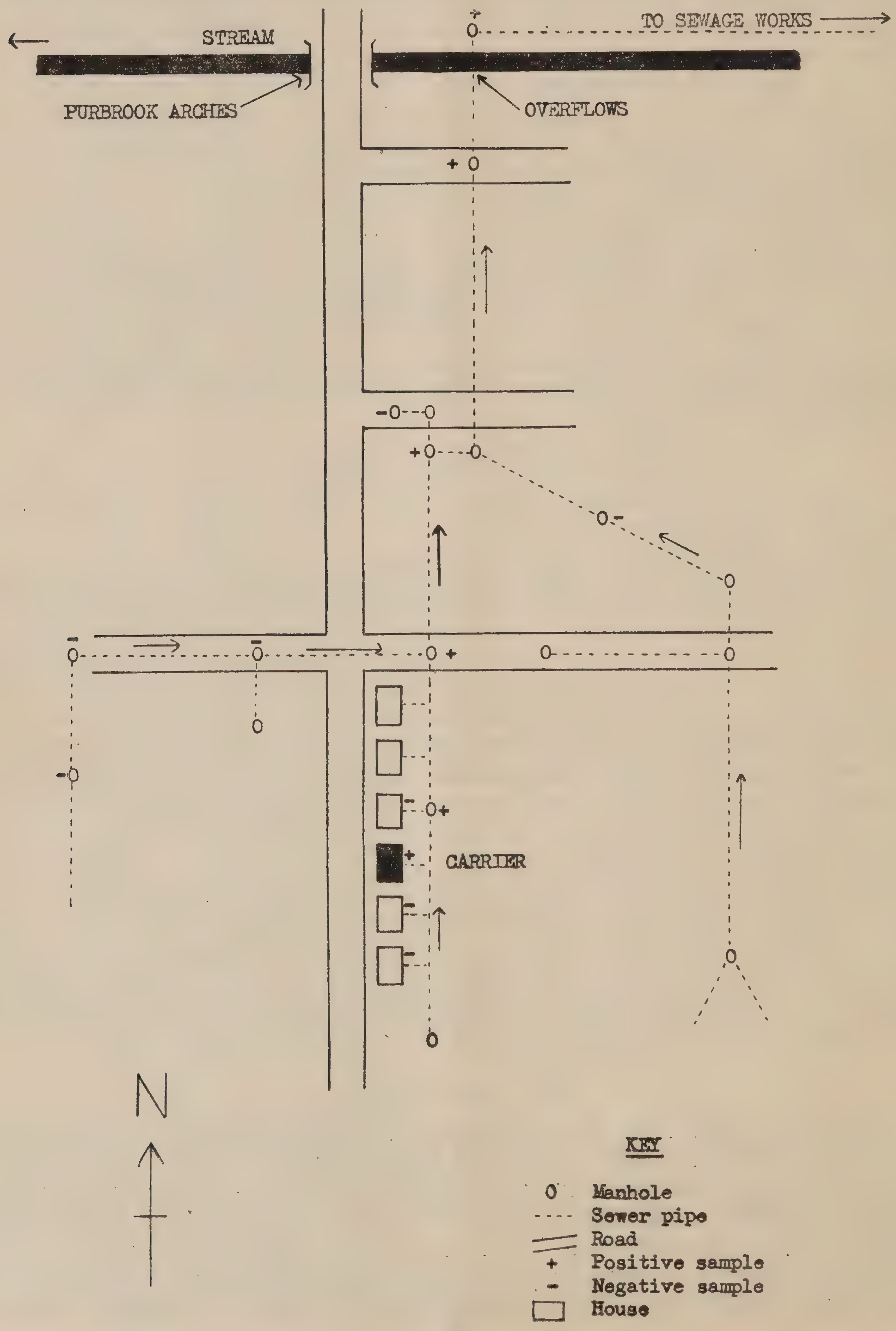
As has been mentioned above, this branch of the Wallington River rises at Crookhorn Farm, and then about half a mile further down it runs past the residential area of Purbrook, crossing under the London—Portsmouth Road at Purbrook Arches. The upper quarter of a mile or so appeared to be reasonably free from contamination, but in the banks of the stream where it runs parallel with Penjar Avenue, Purbrook, the open ends of several large overflow pipes were seen, and signs of contamination became increasingly evident.

These overflows, which connected directly with the Purbrook sewage system, were placed there to relieve the excessive pressure that occurred during heavy rainfall, and as a result large quantities of crude sewage were on occasions discharged directly into the stream. At the time of our visit the weather was dry and the overflows were not in use. Inspection of the remainder of the stream showed no further obvious source of contamination.

FIELD SKETCH OF PURBROOK WITH LOCATION OF CARRIER

House of carrier is shown in black

(This plan is only diagrammatic and does not give the true position of the carrier's house)



In view of these findings it was decided that a number of samples of river water should be collected at intervals over a stretch of about two miles, from the region of Pigeon House Lane Ford up to the source at Crookhorn Farm, in the hope of gaining further information on the origin of the typhoid infection. Thanks to the keenness of the Sanitary Inspectors of Havant and Waterloo U.D. and of Droxford R.D., this was easily arranged, and over a period of about six months many dozens of these samples were received and examined. All of these gave negative results until in January, 1949, after heavy rainfall, *Salmonella typhi* (Vi-phage Type E1) was isolated from water collected on the west side of Purbrook Arches, 75 yards downstream from the sewage overflows. Shortly after this further positive isolations were obtained from the mouths of the overflow pipes, thus confirming our suspicions that the infection was entering the river along with the Purbrook sewage. As the population of Purbrook is about 6,000 it seemed unlikely that the investigation could be carried further and all hopes of tracing the carrier were almost abandoned, when fortunately we came across the description of a novel method which had been used successfully in the detection of paratyphoid carriers in towns by means of sewage examination (Moore, 1948). The chief obstacles in the isolation of *Salmonella typhi* from sewage and the eventual tracing and identification of a carrier arise from the high degree of dilution and mixing to which sewage is subjected during its passage through the sewers, the large number of other organisms which are always present, and the intermittent character of the contamination, which even in favourable circumstances may only occur about once in 24 hours. Fortunately the efficiency of modern selective media got over the first two difficulties, and by the use of Moore's swab method, the third difficulty due to intermittency of contamination was also overcome.

The Swab Method of Sewage Examination

This consists essentially in suspending gauze swabs in sewers at various manholes and intercepting chambers, and leaving them in position for varying periods depending on circumstances. In our first attempts they were allowed to remain for 48 hours, but on Dr. Moore's advice we extended this period to 5 to 7 days, and in each case the results were satisfactory. On removal the swabs, saturated with effluent, were at once immersed in a jar containing 50 ml. of double strength selenite enrichment broth in which they were sent to the laboratory, where they usually arrived about two hours after collection.

Cultural Procedure

When the swabs in selenite were received in the laboratory, 1 ml. of fluid from the jars was added to 10 ml. of single-strength selenite enrichment broth and incubated overnight, as also were the jars with swabs *in situ*. These were all plated out next day on deoxycholate citrate agar and Wilson and Blair plates. Experience soon showed that the best results were obtained from the tubes of single strength selenite, since plates streaked with the incubated fluid from the jars were so overgrown with contaminants as to be almost useless. After discussing this difficulty with Dr. Moore, we adopted the following dilution technique. On arrival in the laboratory the enrichment fluid from the jars containing the swabs was diluted 1/10, 1/100 and 1/1000 with single strength selenite, incubated overnight and then plated. These dilutions, especially the 1/10 and 1/100 strengths, gave such excellent results that no technical trouble was encountered in the isolation of *Salmonella typhi*. Both Wilson and Blair and deoxycholate proved very efficient, and the use of both is strongly recommended for any similar investigation.

Tracing the Carrier

The laboratory procedure having now been standardized, the field investigation was put into the hands of Mr. A. W. R. Turnbull, Chief Sanitary Inspector, and Mr. L. H. Hayward, Additional Sanitary Inspector, Havant and Waterloo U.D. Thanks to their keenness and expert knowledge of the Purbrook sewage system, they were able to place swabs at various sewer junction manholes and in this way rapidly to narrow the infection down to a row of houses. Further swabs lowered into the intercepting chambers of these houses localized the infection to one house situated about half a mile from the river (see sketch).

Only two people were living in the house at the time and the situation was fully explained to them both. Thanks to their co-operation specimens were readily provided, and from the stools of one an almost pure culture of *Salmonella typhi* Vi-phage Type E1 was isolated. This person, then aged over 70 years, had resided for many years in the Middle and Far East. No definite history of typhoid fever could be obtained, though it was suggested that he might have acquired the infection some 60 years previously whilst domiciled in Portsmouth.

Notes on the use of Moore's swabs

As the practical application of the swab method of carrier detection was entirely in the hands of the Sanitary Inspectors of Havant and Waterloo U.D., we are indebted to Mr. Turnbull and Mr. Hayward for the following observations.

The use of these gauze pads for obtaining samples of fluid passing through sewers was found to be much cleaner, easier and more satisfactory than filling sample bottles by dipping them in sewage. The pads, secured to a strong nail or ring inside the manhole or intercepting chamber, were generally arranged so that their lower ends were just below the surface of the water, and to one side of the channel. This prevented the gauze from absorbing too much fluid and lessened the chances of choking the sewer. The pads, as was soon discovered, could not be used in an exposed position in the open, as they appeared to provide an irresistible attraction to children, who frequently proceeded to remove them almost as soon as they were placed in position.

Discussion

In this paper we have given an account of the isolation of *Salmonella typhi* from polluted river water, a procedure which is now much simplified by the use of modern enrichment media. In this instance the infection was traced about two miles up stream to a point where on occasions crude sewage discharged into the stream. By use of Moore's swab method the infection was easily followed another half mile along the sewers of a residential district to the drains of an individual house, one of whose inmates was found to be a typhoid carrier.

Although Moore, in his original account of the swab method of isolation, mentioned that it had not proved satisfactory with *Salmonella typhi,* he has now been able to record complete success (Moore 1950)—a finding we fully confirm.

An essential factor in the tracing of a typhoid carrier is the typing of each culture as it is isolated. For this we depended on Dr. A. Felix, F.R.S., Director of the Central Enteric Reference Laboratory and Bureau, whose willing co-operation greatly simplified our problem. It may be noted that the cases of typhoid which occurred in association with the Wallington River in 1944 were of Vi-phage Type A, whilst those of 1948 were of Vi-phage Type E1. The typing was carried out according to the internationally adopted typing scheme of Craigie and Felix (1947). The fact that two different types of *Salmonella typhi* were associated with the river over an interval of four years is not remarkable in view of the high degree of pollution which was present.

In carrying out this investigation we would like gratefully to acknowledge the whole-hearted co-operation we received from the members of various Public Health Departments, and especially we would like to mention Dr. T. E. Roberts, Medical Officer of Health and Mr. W. F. Appleton, Chief Sanitary Inspector, Portsmouth C.B., Dr. S. Chalmers Parry, Medical Officer of Health, Mr. F. Lindley, Chief Sanitary Inspector, Mr. H. W. Potter and Mr. H. L. Wenden, Additional Sanitary Inspectors, Droxford R.D. We would also like again to express our appreciation of the keenness and efficiency shown by Mr. A. W. R. Turnbull, Chief Sanitary Inspector, and Mr. L. H. Hayward, Additional Sanitary Inspector, Havant and Waterloo U.D., who were tireless in their pursuit of the carrier. The field sketch was kindly supplied by Mr. Donald Hunt, Assistant Clerk, Public Health Department, Havant and Waterloo U.D.

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

FACTORS AFFECTING THE EFFICIENCY OF AIRCRAFT DISINSECTISATION PROCEDURES

R. P. Tew, A.R.I.C., W. A. L. David, Ph.D. and J. R. Busvine, D.Sc.

The first requirement of any aircraft disinsectisation procedure, is that it must kill mosquitoes and, preferably, houseflies. There is, at present, no general agreement on the timing of disinsectisation in relation to embarkation; but if passengers and crew are on board, it is also necessary to consider their safety and comfort. The procedure which we have previously recommended (1) (2) was 15 gm. per 1,000 cu. ft. of a solution containing 0·5 per cent. w/w pyrethrins and 3 per cent. w/w DDT. This was quite effective insecticidally, but it has been suggested that passengers might suffer slight discomfort at this dosage. Furthermore, the recommended time of closure was ten minutes and there is evidence that a period longer than three minutes cannot be tolerated in hot climates (3).

Experiments were, therefore, planned to determine whether the rate of treatment and the closure period could be reduced. It was considered that the practice of using caged insects instead of free-flying ones, in the previous tests, might have given unduly high estimates of the dosage necessary under natural conditions. This point was investigated by using free-flying insects in some of the experiments.

The Aerosol Dispensers

The aerosols and dispensers tested are shown in Table 1.

TABLE 1

Source and Number	Type	Insecticide solution percentage composition w/w			Approx. output gm./sec.
		Py	DDT	PB	
CMR 1	L.P.	0·4	3	—	1
CMR 2	L.P.	1·2	2	—	1
CMR 3	L.P.	0·4	2	3	1
CMR 4	L.P.	0·4	—	3	1
Am. MS	H.P.	1·2	2	—	3
Am. IS	H.P.	1·2	2	—	1

The aerosol dispensers and aerosol formulae used in the experiments. CMR 1–4, four dispensers filled by Messrs. Cooper, McDougall & Robertson Ltd. Am.MS and Am.IS American dispensers manufactured by the Milwaukee Sprayer Manufacturing Co., Inc. and Messrs. Innis Spedden Co., respectively, and charged with the formula G.651 required by the U.S. Public Health Service.

Abbreviations: L.P. = low pressure; H.P. = high pressure;
Py = pyrethrins; P.B. = piperonyl butoxide.

Methods

The present report covers two series of tests, one at London Airport, Heathrow, and the other at R.A.E., Farnborough. All the general methods remain the same as those already described (1) and (2), and the housefly (*Musca domestica*) and the yellow-fever mosquito (*Aedes aegypti*) were again used as test insects. A few special circumstances arose in these tests such as the use of free-flying insects at Farnborough, and for this reason the details of the two series of tests will be given separately.

A. *Tests at London Airport, Heathrow*

The tests were carried out on the 13th and 27th July, 1950, in a fully-furnished Argonaut type aircraft, standing on the open tarmac at Heathrow. As usual, the insects were taken down to the Airport and stored in a warmed, humified room overnight. On the second date, a rather high mortality of *Aedes* occurred during the night before the tests ; but very few more died during the following day and all living insects looked normal. As a control, insects were exposed in the ventilated aircraft and then returned to the warmed room. The knock-down of these insects at 10 minutes was very low, but rather a high mortality occurred among the *Aedes* 24 hours later (Table 3).

In these tests all the aircraft cabins were used and the caged insects were arranged in the following positions:—

A. On the pilot's seat in the cockpit.

B. On the baggage shelf in the main cabin.

C. Under the seat near the centre of the main cabin.

D. Behind a window curtain in the rear cabin.

Results

Summaries of the results obtained are presented in Tables 2 and 3. A discussion of these results and the conclusions to be drawn from them is presented in conjunction with those of the trials at Farnborough.

B. *Tests at Royal Aircraft Establishment, Farnborough*

The tests were carried out on the 19th and 26th October, 1950, in a fully-furnished Tudor type 2 Aircraft standing in a hangar. Before each experiment the aircraft was heated to about 25° C. by six low temperature electric heaters standing in the gangway. Only the main cabin and the entrance were used, the volume of these two compartments being 4,020 cu. ft.

In these trials, about 700 free-flying houseflies and 200 free-flying mosquitoes were used, in addition to the usual caged insects. They were confined in the main cabin by covering the doors at each end with mosquito netting. The spray was divided proportionately between the main and entrance cabins. At the end of the exposure period the door at the front end of the main cabin, behind the mosquito netting, and the two exit doors, were opened. As the aircraft was in a hangar, the ventilation was not very rapid, but it was shown that caged controls were unaffected by exposure for ten minutes in the cabin after it had been aired for one hour.

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* There is an error in the previous reports. The diameter of the insect exposure cages was always 3½ inches, not 3 inches as stated.

TABLE 2

Summary of comparative tests of Aerosols at London Airport in July, 1950

Expt. No.	Aerosol dispenser	Functioning time (sec. per 1,000 cu. ft.)	Amounts dispersed per 1,000 cu. ft.			Closure time (mins.)	Average percentages of Insects			
							Knocked down		Killed	
			Total gm.	mgm. Pyrethrins	mgm. DDT		*Aedes*	*Musca*	*Aedes*	*Musca*
1 ...	Am. MS	1·6	7·5	90	150	3	2	0	71	6
2 ...	CMR 1	15	16·1	64	483	3	48	24	98	30
3 ...	CMR 2	5	5·95	71	119	3	37	38	85	21
4 ...	Am. MS	1·6	6·4	77	128	5	2	0	84	24
5 ...	CMR 1	15	13·6	55	408	5	65	79	100	85
6 ...	CMR 2	5	5·6	67	112	5	62	91	100	63
7 ...	Am. IS	5	6·5	78	130	3	56	35	85	20
8 ...	CMR 1	15	16·1	64	483	3	20	49	83	73
9 ...	Am. IS	5	5·3	64	106	5	100	56	100	37
10 ...	CMR 1	15	13·7	55	411	5	44	72	100	80
1–6 ...	Controls	—	—	—	—	10	0	0	39	0
7–10 ...	Controls	—	—	—	—	10	6	0	40	0

Temperature: 24–28°C. Relative Humidity: 40–50 per cent.

The caged insects were dispersed as follows in the main cabin:—

A. On the edge of an upturned bunk—front, starboard.
B. Behind window curtain—front-half, port.
C. On the floor under the edge of a seat—middle, starboard.
D. On a rear seat—port.

The free-flying insects were released in the aircraft 20 to 30 minutes before spraying, to allow them time to distribute themselves. No doubt some of them chose inaccessible resting places.

The spray was released as usual and the doors kept closed for 5 minutes subsequently. Knock-down counts of free and caged insects were made after a further 5 minutes. The remaining free-flying insects were collected from 20 to 30 minutes after spraying, and a further 45 minutes ventilation time was allowed before the next test.

It is estimated that about 40-50 per cent. of the flies released were recovered and some 25 per cent. of the mosquitoes. These figures are only approximate.

Results

Summaries of the results obtained are given in Tables 4 and 5.

TABLE 3

Individual results according to the position of the caged insects in the aircraft.

Expt. No.	Average percentages of insects affected in different positions (in each square *Aedes–Musca*)							
	Knocked down				Killed			
	A	B	C	D	A	B	C	D
1	0 0	3 0	4 0	0 0	100 13	41 0	96 7	47 5
2	100 65	60 0	18 32	13 0	100 95	100 5	100 12	91 5
3	100 100	3 3	6 8	40 42	100 83	41 0	100 0	100 0
4	6 0	0 0	0 0	0 0	100 80	59 0	96 17	80 0
5	100 100	65 45	72 94	24 —	100 100	100 57	100 98	100 —
6	100 100	86 97	0 75	62 —	100 100	100 85	100 5	100 —
7	100 86	17 27	24 7	83 18	100 64	69 0	71 5	100 12
8	7 85	21 64	39 48	9 0	100	97	92	3
9	— 61	100 62	— 38	100 64	— 49	100 27	— 3	100 70
10	— 100	77 96	— 24	10 68	— 100	100 100	— 21	100 100

TABLE 4

Summary of comparative tests with aerosols at Farnborough on October 19th, 1950

Aerosol dispenser	Functioning time sec./1,000 cu. ft.	Total gm.	Py. mg.	DDT mg.	P.B. mg.	Average percentage of							
						Aedes				Musca			
						Caged		Free		Caged		Free	
						KD	K	KD	K	KD	K	KD	K
CMR 1	5	5·25	21	157·5	—	57	99·5	100	100	3	25	97	98·5
CMR 3	5	5·25	21	105	157	93·3	100	100	100	87	84	99·5	99·5
CMR 1	10	9·75	39	292·5	—	78·5	100	100	100	58	80	99·5	99·5
Control exposed in aircraft	—	—	—	—	—	—	2 per cent.	—	—	—	0 per cent.	—	—
Control untreated ...	—	—	—	—	—	—	7 per cent.	—	—	—	0 per cent.	—	—

Temperature 25–27°C.
Relative humidity 46–48 per cent.
Closure time 5 mins.
Py.=pyrethrins
P.B.=piperonyl butoxide
K=Kill of insects in cages or of those knocked down in the case of free-flying insects
KD=Knock-down at 10 minutes.

TABLE 5

Summary of comparative tests with aerosols at Farnborough on October 26th, 1950

Aerosol dispenser	Functioning time sec./1,000 cu. ft.	Amounts dispersed per 1,000 cu. ft.				Closure time min.	Average percentage of							
							Aedes				Musca			
							Caged		Free		Caged		Free	
		Total gm.	Py. mg.	DDT mg.	P.B. mg.		KD	K	KD	K	KD	K	KD	K
CMR 1 ...	5	5·25	21	157·5	—	5	5	69	100	100	1	19	96	99·8
CMR 1 ...	5	5·25	21	157·5	—	3	4	46	100	100	1	26	96	99·8
CMR 1V ...	5	5·25	21	—	157·5	5	23	65	100	99·5	20	5	97	100
Control: exposed in aircraft ...	—	—	—	—	—	—	0	0	—	—	0	0	—	—
Control: untreated ...	—	—	—	—	—	—	0	0	—	—	0	0	—	—

Temperature 25–27°C.
Relative humidity 46–48 per cent.
Other abbreviations as in Table 4.

General Results of the Experiments

The results of the two series of experiments just described may be summarised as follows:—

(a) *From the experiments at the London Airport, Heathrow*

(1) The American bomb Am.MS gave very poor knock-down and kill of insects in the two experiments in which it was used. This particular bomb had a very rapid output (about 3 gm. per second) and in order to distribute the dose as evenly as possible throughout the airplane, the operator had almost to run through the cabins. The dose was thrown out in a rather compact cloud which would probably handicap good distribution.

(2) The American bomb Am.IS, the output of which, like the CMR dispenser, is approximately 1 gm./sec., gave a much better performance than the Am.MS.

(3) Against *Aedes* Am.IS gave a better knock-down than CMR 1, but both aerosols gave the same kill.

(4) The formula CMR 1 gives a more reliable kill (particularly of flies, though its knock-down effect is, perhaps, not quite so good), than the Am.IS " G.651 " type. The better kill is, no doubt, due to the higher DDT content. With the G.651 formula there is evidence of revival from knock-down of the houseflies.

(5) The CMR 2 bomb (which contains the same amount of active ingredients as formula G.651) gave about the same knock-down as the earlier CMR 1, when both were used at the same dosage per 1,000 cu. ft. However, the latter formulation gave a better final kill, especially of flies, presumably owing to its higher DDT content.

(6) The very different effects obtained with Am.MS, Am.IS, and CMR 2, all of which contain the same percentages of pyrethrin and DDT, show that the dispenser may be as important as the aerosol formula.

(7) The results obtained in individual cages are set out in Table 3. If these data are examined, it will be seen that the effects of the aerosols vary greatly from point to point. Usually, but not always, the highest knock-down and kill was obtained in the very exposed seat of the cockpit. This variability suggesting uneven distribution of the aerosol is perhaps to be expected with a short exposure to the aerosol during a 3- or 5-minute closure period.

(8) There was, throughout, a higher knock-down and kill with 5 minutes closure than with a 3-minute period.

(b) *From the experiments at Farnborough*

(1) The CMR 1 formula, when the dosage is reduced to the order of 5 gm. per 1,000 cu. ft., instead of the 15 gm. previously recommended, still gives a complete 10 minute knock-down and kill of free-flying mosquitoes, though the knock-down and kill of caged insects is erratic and falls below 100 per cent. In the case of houseflies, the 10 minute knock-down was always better than 96 per cent., and the ultimate kill of insects captured up to the end of the collecting period was 99·5 per cent. or better.

(2) In comparison with the foregoing, when used at 10 gm. per 1,000 cu. ft. the CMR 1 formula is decidedly better against caged flies and somewhat superior in all respects.

(3) The CMR 3 formula, which contains piperonyl butoxide, is markedly superior in knock-down to the CMR 1 formula. In the only case where its killing powers can be judged (caged flies) it is also definitely superior.

(4) With the CMR 1 at 5 gm., the closure period can be reduced from 5 minutes to 3 minutes, without reducing the kill of free-flying mosquitoes below 100 per cent. or free-flying flies below about 99 per cent.

(5) Formula CMR 4 in which the DDT of formula CMR 1 has been replaced entirely by piperonyl butoxide gave promising results. It may be of interest if DDT resistant flies have to be considered, but further tests are required to establish the efficacy of this aerosol.

(6) Free-flying insects are much more easily killed than caged insects.

Discussion

The two series of tests at Farnborough show clearly that previous recommendations were unnessarily cautious, if it is agreed that the results obtained with free-flying insects give the best guide to the dosage necessary in practice. It appears that both the dosage and closure time can be safely reduced.

A word should be said at this point regarding the practice of using caged insects adopted in the earlier tests. It had been thought that the cages represented, to a not unnatural extent, the degree of shelter which insects might find among the very complex fittings and furnishings of an aircraft interior. This is probably a fair assumption in itself, but it overlooks the fact that a caged insect contacted by a sub-lethal, but irritating dose of aerosol, cannot leave its shelter, as a free insect would almost certainly do. An obvious practical advantage of caging the insects, was that it simplifies and reduces the labour of the testing procedure. However, it has proved possible to collect knocked-down insects from aircraft rather more easily than had been anticipated, though the job requires about four people equipped with torches and sucking tubes, if it is to be completed within reasonable time. Much more might be said on the subject of disinsectisation experiments in aircraft. The authors are well aware that the procedures they have adopted are open to various criticisms, but unfortunately there is no opportunity to enlarge upon this topic here.

Recommendations

The following recommendations regarding aircraft disinsectisation may be made as a result of experience gained in the foregoing tests:—

1. Use a low-pressure aerosol dispenser, such as CMR 1, containing 0·4 per cent. pyrethrin and 3 per cent. DDT. Probably CMR 3 would be equally satisfactory, though it has not been so fully tested.

2. Use a dosage of 10 gm. per 1,000 cu. ft.

3. Use a dispenser distributing not more than 1 gm. per second, otherwise it is difficult to get round the plane without overdosing.

4. Keep the doors closed for not less than 5 minutes (or longer if convenient) after all the spray has been applied.

5. Although it has been shown that disinsectisation may be effected at dosages which passengers might not find objectionable, their presence is, in our view, likely to interfere with the distribution of the aerosol apart from providing more sheltering places for insects. We recommend, therefore, treatment after luggage has been loaded but before the passengers embark.

Acknowledgments

We are indebted to Sir H. Whittingham and Dr. Peffers of the B.O.A.C., for kindly making available facilities for our tests at the London Airport and to Group Captain L. S. Snaith and Mr. E. W. Press, who arranged similar facilities at Royal Aircraft Establishment, Farnborough.

We also wish to thank the following who have kindly assisted in planning or carrying out these experiments. Drs. R. A. E. Galley and A. B. P. Page, Miss M. Harrison, Messrs. K. F. Goodwin-Bailey, J. L. Hitchon and Mr. J. N. Holborn.

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MOSQUITO NUISANCES IN LONDON

P. G. Stock, C.B., C.M.G., C.B.E., F.R.C.P., D.P.H. and P. G. Shute, M.B.E., F.R.E.S., Assistant Director, M.R.C. Malaria Reference Laboratory.

During recent years many London boroughs have been troubled with mosquito plagues between the months of June and September and a paper was published in this Bulletin for November, 1949. As complaints continue to be received it is hoped that the present note may help those called upon to deal with any such nuisance.

There are two very closely allied species of mosquito to be found in the Metropolis. Unfortunately, both the larval and adult characters of the two species resemble each other closely and even systemic entomologists cannot always separate them with certainty. At first sight, this may hardly seem of importance to those who have to abate a nuisance caused by one or other of the species involved, but the nature of the problem must be appreciated if effective control measures are to be carried out.

The two species are:—(1) *Culex pipiens* which does not bite man; and (2) *Culex molestus* which is a persistent biter.

Culex pipiens is anautogenous (i.e. the female is unable to lay fertile eggs without a previous blood meal). The species is also eurygamous (i.e. swarming for mating purposes).

As far as is known, *C. pipiens* never feed on man or other mammals, but obtain their blood meals from birds, fowls and frogs. Its larvae occur in cesspools, rain-water barrels, stagnant ditches and in containers of all kinds in and around houses, gardens, allotments and refuse dumps.

At the end of the breeding season (September—October), all the males and the larvae die, but the females seek shelter in cellars and outhouses, where they hibernate until the following spring, when they leave their winter quarters, lay their eggs and start the new season's generation.

Culex molestus is autogenous (i.e. the female can lay fertile eggs without a previous blood meal). It is stenogamous (i.e. males and females can mate without the act of swarming). *C. molestus* feeds readily on man. The larvae have on rare occasions been found in above-ground water containers, but prefer breeding places which are dark, especially collections of water underground. Larvae are often present in enormous numbers in water only a few inches deep. The complete life history of *C. molestus* is not yet known, but it is believed to be unable to hibernate in either the larvae or adult form. Observations, however, indicate that breeding continues throughout the winter and that larvae develop uninterruptedly in water in basements and, as was found during the war, in the deep shelters where the temperature seldom fell below 60°F. The inverts of the London Underground Railways where the temperature did not vary greatly formed ideal breeding grounds.

The above outlines our present knowledge of the habits of the two species of mosquito indigenous to London and some other large cities in England, and is summarised in the following table:—

Culex Pipiens

Anautongenous Unable to lay fertile eggs without a previous blood meal.	Never feeds on man.	Larvae present in most artificial water containers as well as in stagnant water, ditches, etc.	Pass the winter as female adults hibernating in cold shelters.	*Eurygamous* Swarming occurs for mating purposes.

Culex Molestus

Autogenous Able to lay fertile eggs without a previous blood meal.	Feeds on man.	Larvae present in stagnant water in semi or complete darkness; usually below ground.	Breeding continues throughout the winter but the adults do not leave the warmth of their breeding grounds.	*Stenogamous* Mating occurs without swarming.

In order to destroy or control an insect pest it is essential to know as much as possible of its life history so that the control measures can be directed against the weakest link in the biological chain. On occasions much energy, money and materials have been expended without appreciably reducing the nuisance. *The reason is that the control measures have been directed against the mosquito larvae found breeding in collections of water above ground, which are those of the non-biting Culex pipiens.* By such measures alone the real culprit, the blood-sucking *C. molestus,* would escape destruction because its breeding grounds would not have been located.

Where there is a mosquito nuisance in London or other large cities, a thorough search must be made for the active breeding grounds, which in the case of *C. molestus,* there is reason to believe, are nearly always below ground level. *Failure to control this species is generally due to failure to locate the breeding grounds.* These may be disused cellars, collections of stagnant water in underground passages, old underground air raid shelters which have become water-logged, and, only occasionally, collections of water in semi dark positions above ground.

When the breeding grounds are located, extermination is very easy. In most cases the stagnant water can be got rid of and the places kept dry. Where breeding occurs in sumps and other deep waters, a bag of sawdust soaked in oil can be lowered into the water. This will prevent mosquitoes breeding for at least two months. If the breeding grounds therefore cannot be dried up, freshly prepared oil-soaked sawdust should be added about once every two months. Local authorities will, of course, be justified in destroying mosquito larvae wherever they may be found in collections of water above ground, but, and *this is the object of this note,* it will not solve the problem of a mosquito nuisance caused by *C. molestus.* This will only be achieved by locating the breeding grounds which are almost always below ground level.

Summary

1. Should a mosquito nuisance arise the first step is to identify the species responsible.

Specimens for identification, especially female adults, alive or dead, may be sent to the M.R.C. Malaria Reference Laboratory, Horton Hospital, Epsom. The mosquitoes should be packed in narrow test tubes between plugs of cotton wool so that the insects will arrive in a good state of preservation.

2. When the nuisance is known to be due to *C. molestus,* a thorough search must be made for underground breeding places. Usually the principal breeding places are within 100 yards of the area where the adult mosquitoes are in the greatest density.

3. Although these mosquitoes are not troublesome in cold weather, breeding is continuous somewhere below ground ; surveys and anti-larval operations should be carried out *before* the approach of warm weather in order to *prevent* a nuisance occurring.

4. Where possible all active breeding grounds should be dried up, but if this is impracticable, other anti-larval measures should be undertaken, e.g. the use of sawdust soaked in oil.

AN OUTBREAK OF COWPOX AT MARSH GIBBON, BUCKS.

E. Donaldson O.B.E., M.D., D.P.H., Ministry of Health, and
D. H. Waldron, O.B.E., M.D., D.P.H., D.T.M. and H., Medical Officer of Health, Buckingham Rural District Council.

Outbreaks of cowpox are probably far from uncommon, although seldom reported, the disease not being notifiable and usually trivial. In the last 50 years eight outbreaks have been reported in the literature, three of which were investigated by the medical staff of the Local Government Board and the Ministry of Health.

In the 32nd Annual Report of the Medical Officer of the Local Government Board, the late Dr. S. Monckton Copeman described in detail an outbreak near Reigate in 1902, in which 80 cows and five out of six cowmen were affected. The virus appeared to have been introduced from a farm at Merstham, where the whole herd and two cowmen had been affected. Dr. Copeman suspected that there might be some connection with an outbreak of smallpox at Croydon, but this could not be proved.

In 1909 the late Dr. Reece* investigated an outbreak in Somersetshire, which occurred in a number of farms spread over about 100 square miles of country. Dr. Reece found that the farms were all on the route taken by a drover from Bristol, where smallpox happened to be prevalent at the time. He, like Dr. Copeman, was unable to prove any connection, but thought that there was some possibility of the disease having been spread in that way.

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* Reece, R. J., Proc. R. Soc. Med., XV, (Epidem.) 13.

Outbreaks of cowpox in human beings all seem to follow much the same pattern. A number of cattle on a farm develop the disease, and some of the milkers, who are unvaccinated, or were only vaccinated in infancy, become infected—nearly always on the hands, but occasionally, as in this Marsh Gibbon outbreak, on the face.

As the disease undoubtedly confers on the sufferers immunity for a time against *smallpox*, it might be considered rather a blessing than a curse. It is, however, obviously a clumsy and unscientific method of obtaining immunity.

From the point of view of preventive medicine, cowpox is of importance owing to its resemblance to very mild smallpox. Moreover, a case of smallpox in a partially immune person might be wrongly diagnosed as cowpox and so set an epidemic going before its true nature was discovered.

Yet another possibility is that a patient previously immunised by cowpox and suffering from some other disease resembling smallpox, might fail to react to vaccination for diagnostic purposes, and thus cause some alarm.

On 21st November, 1949, Dr. Waldron, medical officer of health, Buckingham Rural District, telephoned that a case of cowpox at a farm in the parish of Marsh Gibbon had been reported to him. He asked whether the milk supply should be stopped, and was advised that this was unnecessary, more especially as it was sold to a firm in Aylesbury, by whom it was heat-treated before being tinned. The only milk consumed untreated was a small quantity used on the farm. The farmer was advised to boil this.

As the disease is not notifiable, there had been some delay before the medical officer of health received information of the occurrence. He only heard of it by chance a week after the patient's admission to hospital.

On 11th November, G.K., the foreman on the farm, had consulted his doctor, Dr. Lewis of Steeple Claydon, complaining of vesicles on his hands. Dr. Lewis suspected cowpox, and referred him to the Radcliffe Infirmary at Oxford, whence he was transferred to the Slade Infectious Diseases Hospital, where the diagnosis was confirmed.

When Dr. Waldron visited the farm on 21st November, he found that the farmer, Mr. P., and his brother-in-law, Mr. T., were also suffering from cowpox.

The farm was visited again on 23rd November, by Dr. Waldron, Dr. Donaldson of the Ministry of Health, and Major McIlroy of the County Veterinary Department. Mr. P., was out but Mr. T., was seen, and was found to have a scabbing pustule on the dorsum of his left thumb and a similar lesion on his nose. All the eighteen cows were found to be infected with cowpox.

The worst of the human cases was G.K. On admission to the Slade Hospital he had lesions in all stages on his hands, and, although his temperature was only 99°, he was ill and looked toxaemic. His epitrochlear and axillary glands were enlarged. He had never been vaccinated.

Mr. T., had obviously nearly recovered. So far as one could judge from questioning him, he had not been nearly so ill as G.K. He had been vaccinated in infancy.

Crusts from a lesion on a cow's teat, and some sero-pus from one of Mr. T's lesions, were sent to the Virus Reference Laboratory at Colindale. The former gave negative results—probably because the teats had been treated with an antiseptic ; but the latter was found to be positive for cowpox.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, DECEMBER, 1950

Issued from the General Register Office, Somerset House, W.C.2.

	Dec. 2nd	Dec. 9th	Dec. 16th	Dec. 23rd	Dec. 30th	Average weekly figures for Dec., 1949
Scarlet Fever	1,435	1,266	1,354	1,111	1,026	2,639
Whooping Cough	4,458	4,444	4,523	3,735	3,690	963
Diphtheria	45	49	50	46	41	90
Measles, excluding Rubella ...	12,323	14,706	14,287	14,392	17,468	1,753
Acute Pneumonia	497	574	686	648	962	721
Meningococcal Infection ...	32	33	29	31	34	30
Acute Poliomyelitis (Paralytic)	90	73	82	88	36	97
,, ,, (Non-paralytic)	38	31	26	15	17	7
Ophthalmia Neonatorum ...	33	34	23	37	21	34
Puerperal Pyrexia and Puerperal Sepsis	94	89	66	74	38	64
Dysentery	856	934	1,242	1,024	691	181
Paratyphoid	8	2	5	3	6	5
Typhoid	1	6	2	5	—	10
Smallpox...	—	—	—	—	4	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Mycological Reference Laboratory

Owing to the resignation at the end of 1950 of Dr. R. W. Riddell, Dr. Jacqueline Walker has been appointed Acting Director of the laboratory. Specimens for examination for pathogenic fungi should be addressed to her at the Mycological Reference Laboratory, London School of Hygiene & Tropical Medicine, Keppel Street, London, W.C.1.

AN EXPLOSIVE OUTBREAK OF PARATYPHOID B FEVER DUE TO Vi-PHAGE TYPE "TAUNTON"

PART I.—EPIDEMIOLOGY

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Central Public Health Laboratory, Colindale,
and
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Originally the number of Vi-phage types and sub-types of *Salm. paratyphi B* known to be indigenous to Britain was four (Felix and Callow, 1943). Subsequent intensive investigation of those strains that remained untypable according to the original typing scheme led to the recognition of a further six Vi-phage types and sub-types, and the routine Vi-phage typing of *Salm. paratyphi B* is now carried out according to this extended scheme (Felix and Callow, personal communication). In the spring of 1948 the records of the Central Enteric Reference Laboratory and Bureau showed an increase in the number of cases of paratyphoid fever due to a strain of *Salm. paratyphi B* which had been provisionally labelled Vi-phage Type "Taunton". This strain was the cause of an explosive outbreak of enteric fever in Eastbourne C.B. during July and August, 1948, and in answer to a request for assistance by the Medical Officer of Health, a temporary laboratory of the Public Health Laboratory Service was set up in the town on 2nd August. The outbreak comprised 50 confirmed cases; 46 were primary, 3 were secondary and one might have been primary or secondary. That cases of enteric fever were occurring was suspected on 25th July, but subsequent inquiry revealed that the first patient developed symptoms on 16th July and that in the following five days 32 persons were affected; the rest of the primary cases developed symptoms between 22nd and 29th July. The secondary cases, which were in home contacts of primary cases, occurred on 11th, 15th and 20th August; the case which might have been primary or secondary was in a patient who became ill on 8th August and who was a home contact of a confirmed case.

During the first two weeks of July the weather was mainly dry and sunny and the mean atmospheric temperatures ranged from 54° F. (minimum) to 67° F. (maximum).

As 14 of the first 15 cases gave a history of having eaten cream cakes from a certain bakery in the town, investigation was first concentrated on the bakery, the directors of which gave their full co-operation. The establishment consisted of a central bakehouse and eight branch shops in different parts of the town. No bread or confectionery was sold directly from the bakehouse, although roundsmen collected bread (not cakes) from there and sold it on their rounds. Cases occurred in customers at all the branch shops, but not in the inhabitants of one area of the town where there was no branch shop, but which was supplied with bread by van.

The Bakery

Six months before the outbreak extensive modernization had been begun at the bakehouse and at the time of the outbreak was only partly completed. At one time a portion of the roof was removed and a temporary covering substituted. Additional drainage work was carried out, and on 12th July a temporary blockage occurred in the drainage manhole. To facilitate the progress of the work the old sanitary annexe consisting of W.C., urinal and lavatory basin with hot and cold water had to be demolished and a change-over to the new accommodation had to be made before the hot water supply was connected to the basins.

When the bakehouse was visited, it was found that the various sections making bread, cakes, and confectionery were crowded into one large room. No hot water was available for hand washing, and no towels were seen. The washing-up tanks had been removed and, as replacements had been late in arriving, the washing up of all utensils, including the basins in which synthetic cream for the cakes was whipped, was being carried out in one large tank. Infection could have spread very readily from one section to another by the washing-up tank. The Savoy bag (a permeable canvas container) from which the cream was squeezed into the cakes was also washed in this tank, although it was stated that it was often boiled after being washed.

It was ascertained that all items of confectionery were baked, with the exception of the cream fillings and a small amount of a margarine-sugar filling.

Staff of the Bakery

Thirty-three employees worked at the central bakehouse, 17 of whom prepared or handled at some stage the products of the confectionery department. The 17 confectioners were bled at the first visit and their sera examined for the presence of agglutinins. Serum from one of the directors of the bakery, who had worked at the confectionery table early in July for the first time for five years, agglutinated *Salm. paratyphi B* H Phase 1 (type) to a titre of 1/80 trace. He had no history of inoculation. Similar titres were found in other confectioners who had a history of inoculation, but these were not considered significant as the titres to *Salm. typhi* H and *Salm. paratyphi A* H were also raised. None of the confectioners gave a history of recent suspicious illness except the director, who had suffered from dyspepsia for eight years, and whose daughter had, about a year previously, contracted an undiagnosed illness with abdominal symptoms. In view of this history and of the suggestive H agglutination titre with *Salm. paratyphi B*, eight specimens of stool, three samples of urine and one sample of duodenal contents were obtained from the director. All were negative for pathogenic organisms. A sample of serum from his daughter showed no agglutinins to *Salm. paratyphi B*. Blood samples were later obtained from the 16 other men who worked in the bakery, from 15 roundsmen, and from 23 builders' workmen who had been in the bakery during July. With the exception of the serum from one roundsman (T.M.) and one workman who denied T.A.B. inoculation, no significant titres were obtained. The roundsman was the father of one of the early cases and had himself eaten cream cakes during July.

Serological examinations carried out on the director of the bakery, on the roundsman, and on the workman are shown in Table 1.

TABLE 1

Results of agglutination tests on three possible originators of the outbreak

Description	Date	T H	A H	B H Phase 1	B H Phase 2	B O	B Vi
Director ...	August 1948	—	—	1/80 trace	—	—	=
	December 1948	n.t.	n.t.	—	—	—	=
Roundsman ...	9. 8.48	—	—	—	1/1280	—	=
	18. 8.48	—	—	—	1/1280	—	=
	6. 9.48	n.t.	n.t.	—	1/640	—	n.t.
	29. 9.48	n.t.	n.t.	—	1/200	—	=
	26.11.48	n.t.	n.t.	—	1/50	—	=
Workman ...	August 1948	—	—	—	1/80 trace	—	n.t.

Explanation of Table

T H = *Salm. typhi* H.
A H = *Salm. paratyphi A* H
B H = *Salm. paratyphi B* H
B O = *Salm. paratyphi B* O
B Vi = *Salm. paratyphi B* Vi
n.t. = not tested.
— = negative at dilutions of 1/20 and above.
= = tests carried out by Dr. A. Felix and found negative at dilutions of 1/5 and above.

The first two specimens of stool obtained from the roundsman were found to be negative, but the third yielded *Salm. paratyphi B* Vi-phage Type "Taunton". Ten subsequent specimens of faeces, one specimen of duodenal contents and three specimens of urine were negative. He emphatically denied any history of abdominal or other illness during the previous six months. It was considered that this roundsman was a temporary symptomless excreter who was probably infected at the same time as his daughter. No positive specimens of stool were obtained from three out of four other members of this family, all of whom had regularly eaten cream cakes from the bakery.

Three stools obtained from the workman listed in Table 1 were negative on culture.

Two bakers who were employed in the bakery early in July had left for other employment before the outbreak occurred. One had gone to Grimsby and one to Australia. Both were traced and specimens of sera, faeces and urine were obtained and examined by the appropriate health authorities. All these specimens were reported negative.

Immediately the full results of the serological screening became known, an attempt was made to obtain three specimens of stool from all the bakery employees, but further investigations now met with resistance from several of the employees, who however submitted to rectal swabbing. All the rectal swabs were negative, as were specimens of urine which were obtained with little further difficulty except from four employees who refused to co-operate any longer.

Synthetic Cream

Of the 46 persons primarily infected, 42 gave a firm history of having eaten the suspected cream cakes during July, while the remaining 4 stated that they had very probably eaten them. Ten ate the cakes on one occasion only during

July—one on 6th, three on 7th, one on 8th, two on 13th, one on 14th, one on 16th and one on 17th. Some 15,000 cream cakes were produced weekly by the four large bakeries in the town, and many smaller shops, cafés, etc., also sold cream confectionery. The suspected bakery produced only 1,800 of the total number.

The cream was stored in sealed containers in a refrigerator in the bakehouse and was used within 2 days of delivery. Each morning the requisite amount of cream was taken from the refrigerator and mechanically whipped while the sponge was being baked. The sponge was filled as soon as it was sufficiently cool, and the cakes were distributed to the shops by 10 or 11 a.m. They were all sold by lunch-time and it was thought that most of them were eaten at tea-time on the same day ; customers were told that the cakes should not be kept overnight. Culture of opened and unopened tins of synthetic cream found in the bakery gave negative results.

The synthetic cream for the fillings was supplied from three sources, one local and two in the London area. The local factory was visited and found to be conducted on hygienic lines ; bacteriological reports on all batches of cream had been satisfactory for at least 18 months, and sera from members of the staff were examined annually. One of the two factories in the London area was also visited, as cream from this factory was suspected in connection with another outbreak due to *Salm. paratyphi B* Vi-phage Type " Taunton ", but stools, urine and serum samples from members of the factory staff were examined with negative results. The third factory was not investigated as no other outbreak was traced to its products, which had been widely distributed.

Other Epidemiological Investigations

Milk was excluded as a source of infection, for although 72 per cent. of the patients obtained milk from one dairy, 78 per cent. of the population were supplied from it, and five of the patients obtained all their milk from other sources.

There was no evidence to incriminate the water supply.

Ice-cream was also excluded, as no common source was discovered, and it was ascertained that five of the patients had eaten no ice-cream during July.

Cultures taken from other materials in the bakery, viz., marshmallow powder, buttercream emulsions, scrapings from refrigerators, liquid cracked eggs, slime from the washing-up tank, and scrapings from the drain, were negative.

The Cases

Fifteen of the patients were males and 35 were females. Twenty-one cases were in children under 6 years of age (See Table 2.)

Salm. paratyphi B Vi-phage Type " Taunton " was isolated from all the clinical cases except one who yielded an untypable culture, although two other members of the same family yielded cultures that were readily typed as belonging to Vi-phage Type " Taunton ".

Four of the patients were infected while on holiday in Eastbourne, and returned home before symptoms developed ; one case occurred in the adjoining rural area and one local resident became ill while on holiday in Jersey. In each of these six instances the infecting organism was also found to belong to Vi-phage Type " Taunton ".

Forty-one of the 44 patients treated locally presented a typical picture of enteric fever, with continued fever, prostration, headache and constipation. The three secondary cases were very mild, and in the absence of an epidemic they might easily have escaped diagnosis. In view of the epidemiological importance of such cases, their histories are given below:—

1. H.P., male, aged 3 years, was perfectly well until the morning of August 11th when his temperature was found to be 101° F. By 3 p.m. on the same day he was well, and remained so until August 18th when he developed diarrhoea; this lasted for 3 days, during which time he passed 20 motions daily. On the evening of August 18th his temperature was 103° F., but it returned to normal on August 20th.

2. E.M., female, aged 47 years, a contact of an early case, complained on August 20th of diarrhoea; this lasted until the morning of August 21st. During this time she passed eight stools, and complained also of headache and lassitude. She recovered on the evening of August 21st and remained well (although constipated) until August 30th. Three stools were examined during this intervening period and were negative. On September 1st, diarrhoea began again and persisted for 3–4 days, followed by 2–3 days of abdominal discomfort.

3. R.H.P., male, aged 42 years, suffered from abdominal discomfort on August 15th, and felt unwell on the 16th but continued at work. On August 17th he still felt ill, had a temperature of 101° F. and developed diarrhoea, which lasted for about 36 hours.

TABLE 2

Age and sex distribution of cases

Age (in years)	Cases		
	Male	Female	Male and Female
0—	0	3	3
2—	2	7	9
4—	4	5	9
6—	2	1	3
8—	0	0	0
10—	1	2	3
12—	0	1	1
14—	0	0	0
16—	0	1	1
18—	0	0	0
20—	0	5	5
30—	3	6	9
40—	1	0	1
50—	2	1	3
60+	0	3	3
Total ...	15	35	50

The results of serological and cultural examinations carried out on these three mild cases are shown in Table 3.

The serological reactions in the typical cases were by contrast quite straightforward, and in each of the eighteen cases from which sera were obtained after the first week of infection, adequate serological evidence of enteric infection was present.

Discussion

The epidemiological evidence incriminating the cream sponge cakes as the vehicle of infection was considered conclusive. During baking, the centre of the sponge reached a temperature of nearly 100°C., and it is therefore almost certain that infection was conveyed by the unheated cream fillings.

The method by which the cream became infected was not determined. There had been ample opportunity for it to become infected directly or indirectly from the hands of a missed case among the bakery staff, particularly by way of the washing-up tank, which was used by almost the entire staff for general washing up, including the cleaning of bowls used for the cream. Once infection of a cream-bowl had occurred it could have spread to other mixes either by way of the washing-up tank or of the Savoy bag. Other possibilities are that infection may have been conveyed to the cream from some other foodstuff imported into the bakery ; that an isolated batch of cream may have been infected before receipt at the bakery ; or that infection may have arisen from the blocked drain, specimens from which, if examined by the method subsequently described by Moore (1948), might have thrown further light on this source.

TABLE 3

Results of serological and cultural investigations in three mild cases

CASE and date of onset of symptoms	Serological Examinations				Cultural Examinations		
	Date	B H Phase 1	B H Phase 2	B O	Dates, Stools +	Dates, Stools —	Blood Culture
H.P. ... 11/8 } ? 18/8 }	24/8	—	—*	—*	24/8, 3/9, 13/9, 15/9, 17/9, 20/9, 27/9, 4/10, 11/10, 18/10, 25/10, 1/11, 9/11	10/11, 20/11, 23/11	n.t.
E.M. ... 20/8 ...	30/8 9/9	— —	— —	— —	3/9, 13/9, 15/9, 17/9, 20/9, 29/9, 4/10, 11/10, 15/10, 18/10, 25/10, 1/11, 9/11, 16/11, 23/11, 11/12, 1/1/49, 4/1, 11/1	24/8, 26/8, 30/8, 30/11, 4/12, 7/12, 18/1/49, 25/1, 31/1, 1/2, 7/2, 8/2, 12/2, 16/2, 22/2	n.t.
R.H.P.... 15/8 ...	24/8 31/8	— —	1/20 (trace) 1/20 (trace)	1/20 (trace) 1/20 (trace)	17/8, 24/8	3/9, 6/9, 7/9, 8/9	negative on 18/8

Explanation of Table 3

Symbols used here have the same meaning as in Table 1.

—* = Insufficient serum for testing at 1/20, negative at dilutions of 1/40 and above.

Dates, stools + = Dates of receipt of stools from which *Salm. paratyphi B* was isolated.

Dates, stools — = Dates of receipt of stools from which no pathogens were isolated.

The following conclusions may be drawn from the investigation.

(1) A saving of time could have been effected if apparatus and media earmarked for emergencies had been held ready in a regional Public Health Laboratory Service depot. On this occasion, owing to August Bank Holiday, 48 hours were lost while the necessary equipment was being selected from stores and from routine stock.

(2) The necessity for bacteriological examinations of all possible carriers should be strongly emphasized at the outset of any inquiry. In this particular outbreak, serological screening of bakery employees was undertaken before stool cultures, and the men obtained a false idea that a single blood test would give conclusive results. When specimens of stool were subsequently requested, a number refused on the ground that submission to blood examination should have been sufficient. We consider that, if at the outset it had been categorically stated that at least three specimens of stool would be required from each of the 33 employees, these specimens would have been obtained.

(3) Though serological evidence of recent infection is commonly obtained in typical clinical cases, it may be absent in mild cases. Since the typical case may also be brought under suspicion by the quicker method of history taking, we feel that when search is begun for the human source of infection in this type of outbreak undue attention should not, at the outset, be given to " serological screening ", and that serological examinations should be carried out supplementary to stool cultures, which should be performed on *all* suspected carriers as soon as possible. The value of serological examination is illustrated in the roundsman, and also in the director of the bakery, in whom retrospective serological evidence suggestive of fairly recent paratyphoid infection was subsequently obtained by virtue of the declining agglutinin titres.

Thanks are due to Dr. A. G. Shera, Consultant Pathologist, Princess Alice Memorial Hospital, Eastbourne, for his helpful co-operation, and to Dr. A. Felix, F.R.S., who gave a great deal of help and advice during the investigation.

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PART II. BACTERIOLOGY

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Laboratory investigations included cultures and agglutination tests. Cultures were carried out on stools and rectal swabs, on blood, urine and duodenal fluids, and on materials obtained from the bakery. Agglutination tests were performed on suspected cases and carriers.

Cultural Examinations

Stool and urine cultures were carried out on suspected cases, on carriers, and on convalescents. The criterion of cure was taken as six consecutive negative stool cultures during convalescence and three consecutive negative urine cultures taken concurrently with the last three stools. In children and males unlikely to handle food the criteria were relaxed to three consecutive negative stools and one urine.

Cultural examinations other than stool cultures will be dismissed in one paragraph. *Blood cultures* were taken from fourteen bacteriologically negative cases and from three positive cases. Two of the latter three cultures were positive ; the negative culture was obtained from a mild case, R.H.P., on the third day of his illness. *Urine cultures* were all negative with the exception of four specimens of urine received from two convalescent cases between the sixth and eighth weeks of clinical infection. Stools taken over the same period from both these cases were also positive. No positive cultures were obtained from *rectal swabs, duodenal fluids,* or from materials obtained from *the bakery.*

Stool Cultures

The routine selective media used were Wilson and Blair's medium (Difco), Hynes' (1942) modification of Leifson's medium, and tetrathionate broth for enrichment culture. Six ml. of freshly prepared 8 per cent. ferrous sulphate per litre were added to reconstituted Wilson and Blair's medium. Though this quantity of ferrous ion is in excess of the amount advocated by Hobbs

(1943) for the isolation of *Salm. typhi*, the medium was reasonably selective and gave excellent blackening with *Salm. paratyphi B* and *Salm. typhimurium*. Wilson and Blair plates were poured daily and used on the same day, or after overnight refrigeration. The tetrathionate broth was a "balanced" tetrathionate corresponding to the medium A described by Rolfe (1946), with the exception that brilliant green was omitted. This medium approximates to number 55 (*c*) described by Knox, Gell and Pollock (1943).

Stools were plated direct, and after overnight tetrathionate enrichment, on to both solid media, except when the direct platings were clearly positive after overnight incubation. All plates negative at 24 hours were replaced in the incubator for a further examination on the following day. A plate microscope proved a useful accessory, particularly in the examination of Leifson plates, on which colonies of this strain of *Salm. paratyphi B* were rather effuse, with rough margins, and had a very characteristic striation with light markings throughout the substance of the colonies. This appearance was present in every colony of *Salm. paratyphi B* on Leifson's medium that was confirmed as such, and was absent from all but a small minority of *Proteus* colonies. On several occasions isolated colonies of *Salm. paratyphi B* could confidently be picked out even when surrounded with and overlapped by colonies of *Proteus*. This same striated effuse margin was occasionally seen on direct Leifson platings, appearing at some part of the edge of a most unpromising confluent mass of lactose-fermenting bacterial growth during the second day of incubation. Surprisingly, *Salm. paratyphi B* could be isolated in pure culture from these striated marginal growths.

With regard to the relative merits of Wilson and Blair and of Leifson's solid media, our findings were in general agreement with those of Hobbs and Allison (1945). As a rule, if the organism grew at all, it was present on both media, but there were exceptions in favour of each medium, both from direct platings and from tetrathionate enrichment. Thus in ten direct platings, *Salm. paratyphi B* was isolated from Leifson's medium alone in six, and from Wilson and Blair's medium alone in four. In four tetrathionate enrichment platings, there was a 3:1 ratio in favour of Wilson and Blair's medium. In several of these instances numerous colonies of *Salm. paratyphi B* were present on the one positive plate, and it is suggested that these findings and those of other workers (Hobbs and Allison, 1945) cannot wholly be explained on the basis of sampling error. It is possible that the composition of the associated bacterial flora in stools may on occasion be a deciding factor in the relative successes of different selective media.

The value of tetrathionate enrichment was unquestionable; of 82 positive stools, only 35 were positive on direct plating, and it was decided on 1st September to omit direct plating of stools received from convalescent cases. At about the same time it was becoming desirable to close the branch laboratory, which had been set up primarily for the investigation of the outbreak itself, and to seek alternative means of dealing with the clearance tests on convalescents. If a suitable substrate could be found which, when added to faecal material, would suppress the overgrowth of non-pathogens without inhibiting *Salm. paratyphi B*, stools could then be sent by train to the Central Public Health Laboratory at Colindale. The buffered glycerine saline described by Sachs (1939) for dysentery stools was considered, but in view of the results which had been obtained from tetrathionate, the latter was thought worthy of trial for this purpose. Portions of stools from convalescent cases were therefore put up in duplicate in tetrathionate broths, one series being held for 24 hours at room temperature before incubation in the usual way. Of 39 stools positive by normal tetrathionate enrichment, 34 were positive also after preliminary storage at room temperature in tetrathionate. None of the stools negative by normal enrichment technique was plated out after enrichment by the delayed procedure ; had this been done a few more positives might have been obtained, as it is possible that at least part of the difference observed between the two series may have arisen from sampling error in stools containing only a very few pathogenic organisms. The results thus obtained were considered sufficiently good to justify the closure of the branch laboratory on 9th September, 1948, and all subsequent clearance tests were carried out at the Central Public Health Laboratory until December, 1949, when they were again taken over by the author. Portions of stool were transferred to one-ounce screw-cap bottles containing tetrathionate broth warmed to 37°C, which were received at the Central Public Health Laboratory on the same day. The results of stool cultures between 9th September, 1948, and 21st December, 1949, have been supplied by courtesy of the Central Public Health Laboratory, and have been incorporated in this report.

Stool culture was positive in all the notified cases. Information was received of only one case which was not notified and in which there were clinical grounds for a diagnosis of paratyphoid infection. This patient however, Mrs. F., the mother of a confirmed case, had two blood cultures, four stool cultures, and a full serological examination four weeks after the onset of her illness, and all were negative.

From every clinical case except one, *Salm. paratyphi B* Vi-phage type " Taunton " was isolated from the first stool received for culture ; the exception, E.M., a mild case particulars of which have been given in Part 1, had indefinite symptoms which might well have resulted in a too early assessment of the date of onset of her infection.

The results of stool culture have been summarized in the table, in the preparation of which it has been assumed that cases which had successfully fulfilled their respective criteria of cure subsequently remained bacteriologically negative.

At the end of twelve weeks, *Salm. paratyphi B* was still present in the stools of seven cases, two of which, E.M. and H.P. (referred to in Part 1), had been mild. The cessation of excretion of this organism in the stools was generally abrupt ; only two cases could be described as " intermittent excreters ", and each of these had only one " negative period " lasting between two and four weeks.

It is noteworthy that both cases excreting *Salm. paratyphi B* eighteen months after infection (see Table) were females over 70 years of age, and that a third case, which continued to excrete until the 72nd week, was a boy aged four. Rough changes were apparent by the 69th week in the organism being excreted by one of the elderly females and this change was progressive, so that before the end of the second year the organism had become serologically unrecognizable as *Salm. paratyphi B,* earlier loss of O agglutinability being finally followed by loss of H agglutinability.

The history of one case, a woman of 36, was dramatic ; she yielded an unbroken series of twenty-five positive stools up to and including the date on which cholecystectomy was performed (during the 27th week). A negative stool was received on the day following operation, and no further positives were obtained.

SUMMARY OF STOOL CULTURES OVER A PERIOD OF EIGHTEEN MONTHS IN FORTY-FOUR CASES OF PARATYPHOID B INFECTION, SHOWING NUMBERS OF CASES CULTURALLY POSITIVE AT VARIOUS PERIODS OF INFECTION AND CONVALESCENCE

Weekly periods from onset of infection

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th	13th	14th	16th	20th	24th	28th	32nd	36th	40th	44th	48th	52nd	65th	78th
No. of cases culturally positive	9	26	7	10	19	33	21	16	11	9	7	7	6	4	3	4	4	2	1	2	2	2	2	2	2	2
No. of cases "presumed" positive	0	7	25	22	12	1	2	1	0	0	1	0	0	1	1	1	0	1	2	1	0	1	1	1	1	0
No. of cases culturally negative	1	1	1	2	8	9	17	12	13	8	3	2	3	4	4	0	1	2	0	0	0	0	0	0	0	0
No. of cases "presumed" negative	0	0	1	1	1	1	4	14	20	27	33	35	35	35	36	39	39	39	41	41	41	41	41	41	41	42
Total number of cases positive	9	33	32	32	31	34	23	17	11	9	8	7	6	5	4	5	4	3	3	3	2	3	3	3	3	2
Total number of cases negative	1	1	2	3	9	10	21	26	33	35	36	37	38	39	40	39	40	41	41	41	41	41	41	41	41	42
No. of cases in which data were absent	34	10	10	9	4	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
Percentage of cases positive (approximate)...	90	97	94	91	78	77	52	40	25	20	18	16	14	11	9	11	9	7	7	7	5	7	7	7	7	5

Explanation.—During particular weekly periods under review no stools were received for culture from some of the cases; when this occurred the case was shown as "presumed" positive when the last stool received before, and the first stool received after, the period in question were both culturally positive. "Presumed" negatives were deduced by similar inference, but when the preceding and succeeding stools were of different sign, and no stool was received, the case was shown as "data absent".

Agglutination Tests

For routine tests, sera from suspected cases and carriers were put up in doubling dilutions from 1/20—1/320 with suspensions of *Salm. paratyphi B* H (Phase 1), *Salm. paratyphi B* H (Phase 2) (for which a Phase 2 suspension of *Salm. typhi-murium* was used), and *Salm. paratyphi B* O, all supplied by the Standards Laboratory. Whenever the results of these tests might have been attributable to T.A.B. inoculation, suspensions of *Salm. typhi* H and *Salm. paratyphi A* H were also put up for confirmatory evidence. Agglutination tests were carried out according to Dreyer's technique. H agglutinations were read at 2 hours in the 50° C. water-bath, O agglutinations at 4 hours in the same bath and again after being left overnight at 50° C. In special cases referred to later, O agglutination was also put up at 37° C. by the technique of Felix. Except when stated otherwise, the results given below were obtained by Dreyer's technique, and by the use of the first three suspensions referred to above.

The results of the agglutination tests which are given below have for convenience been classified according to the source of the serum, the T.A.B. inoculation history, and the presence or absence of bacteriological confirmation of paratyphoid infection.

Bacteriologically Confirmed Cases

Negative serological findings in three mild cases have been discussed in Part I. Sera from a total of eighteen cases with typical clinical symptomatology were examined during the second and third weeks of infection. Thirteen of these sera were tested before the branch laboratory had been set up, and the results of these tests were kindly supplied by Dr. A. G. Shera of the Princess Alice Memorial Hospital, Eastbourne. H titres of the eighteen cases ranged from 1/500 to 1/100,000, and O titres from nil at 1/20 to 1/500. Although in the five sera which were received at the branch laboratory titres of *B* H Phase 2 agglutinins were lower than *B* H Phase 1 titres, the converse may equally apply, as was strikingly illustrated in sera from the baker's roundsman (see Part I, Table 1). The use of an appropriate *B* H Phase 2 suspension should therefore not be omitted in routine tests of this sort.

Persons in whom Suspicion of Paratyphoid Infection was Based on Clinical Grounds Alone

(1) *Inoculated.*—Sera from five persons were examined between the 5th and 12th days after onset of symptoms. All O titres were less than 1/20. H titres ranged from 1/20 to 1/80.

(2) *Uninoculated.*—Sera from twelve persons were examined during the second, third and fourth weeks from the onset of illness. All O titres, and H titres from ten cases, were less than 1/20. One of the remaining cases had a *B* H Phase 2 titre of 1/160, the Phase 1 titre being less than 1/20; no rise in Phase 2 titre was obtained in a subsequent test, and titres of *Salm. typhi* H and of *Salm. paratyphi A* H were less than 1/20. The other remaining case had *B* H titres of 1/20 both with the Phase 1 and with the Phase 2 suspension.

Suspected Carriers

(Serological investigations on the director of the bakery, a baker's roundsman and a workman have been discussed in Part I and are here omitted.)

(1) *Inoculated.*—Sera from 52 possible carriers were examined. All O titres, and H titres of ten of these were less than 1/20. Forty-two had H titres ranging from 1/20 to 1/320.

(2) *Uninoculated.*—Sera from 19 possible carriers were examined. All H and O titres were less than 1/20 except in one serum which agglutinated *Salm. paratyphi B* O at 1/20.

Agglutination Tests by Felix's Technique

In addition to the technique described above, Felix's technique was used for O agglutination in sera which were of particular epidemiological interest, or in the interpretation of which an element of doubt existed. A serum taken on 18th August from T. M., a baker's roundsman (see Part I, Table 1), though containing no O agglutinins at 1/20 by Dreyer's method, gave a titre of 1/160 to *Salm. paratyphi B* O by Felix's method. The serum was sent to Dr. Felix for *B* Vi agglutination, and a report was received from him in which it was also stated that no *B* O agglutinins were present at 1/50. The serum which had been kept in the refrigerator for four days was accordingly retested, and this time no *B* O agglutinins were detected at 1/20, although the same technique and the same batch of suspension were used as previously. (It should be noted that on the first occasion the serum had been put up for agglutination within two hours of taking the blood.) In order to establish the reason for the discrepancy, a further fresh serum was obtained from this case on 6th September and a portion was heated to 56° C. for ½ hour. The heated and unheated fractions were put up for O agglutination by Felix's technique in parallel; while the *fresh* unheated serum gave a *B* O titre of 1/160 as previously, no *B* O agglutinins were present at 1/20 or over in the *heated* serum. Agglutination produced by the unheated serum was clearly visible to the naked eye in low dilutions, and through a hand lens in the end-point dilution, after shaking up the tubes. (The end-point by the "spot" technique was difficult to determine.) Similar results were obtained with a serum freshly obtained on 31st August from R.H.P., one of the three mild cases referred to in Part I (Table 3). This fresh serum unheated had a titre of 1/160 with *Salm. paratyphi B* O, whilst its heated counterpart was negative at 1/20. A 1/10 dilution of the same unheated serum which had been kept for one night in the refrigerator had a titre of only 1/4, equivalent to 1/40 in the undiluted state. (H and O titres obtained by Dreyer's technique with the same sera have been shown in Tables 1 and 3 of Part I.)

In two bacteriologically confirmed cases, *B* O agglutinins had been demonstrated by Felix's technique *only* when fresh unheated serum was used, and had been absent by Dreyer's technique. The question therefore arose as to whether the influence of some labile factor in the serum was a specific one which could be of diagnostic use, or whether its action was non-specific. When serum from T. M. was obtained freshly on 6th September, it was accordingly at once put up both heated and unheated with suspensions of two other organisms chosen at random, *Sh. shigae* and *Br. abortus.* The titres of the unheated and heated serum fractions with *Sh. shigae* were found to be identical with those which were obtained with *Salm. paratyphi B* O (namely 1/160 and nil at 1/20), whereas with *Br. abortus* both the unheated and the heated fractions were negative at 1/20. These findings, though inconclusive, lend support to a "non-specific hypothesis", since it seems improbable that T. M., who had never been overseas, should have specific agglutinins to *Sh. shigae,* and since the presence in a single serum of antibodies *of equal titre* specific to two unrelated organisms could be due to chance alone, though this finding would be expected with a serum containing a single non-specific antibody reacting with both organisms.

During the limited time available, an unsuccessful attempt was made to determine whether by the addition of guinea-pig complement to heated serum its *B* O titre could be restored to the level in the fresh state. The attempt failed owing to the presence in appreciable titre of O agglutinins to *Salm. paratyphi B* in the complement.

Summary of Parts I and II

An outbreak comprising fifty cases of paratyphoid B fever due to Vi-phage type " Taunton " is described. Infection was spread by the synthetic cream filling of sponge cakes, but the mode of infection of the cream was not ascertained.

Laboratory investigations were carried out on forty-four cases, and stool cultures on these were followed up over a period of eighteen months. The approximate percentages of cases excreting *Salm. paratyphi B* in the stools at one, two, three, six, twelve, and eighteen months after infection were 91, 25, 14, 7, 7 and 5 respectively. The cessation of excretion was in most cases abrupt. In forty-three out of forty-four cases, *Salm. paratyphi B* was isolated from the first stool received for culture ; these included ten cases from which stools were received during the first week, and two from which stools were received within three days of the onset of symptoms.

Tetrathionate broth gave good cultural results both in enrichment culture and when used for the carriage of stools from paratyphoid convalescents to a remote laboratory.

O agglutinations at 37° C. carried out on freshly drawn sera gave higher titres than those observed in the same specimens after storage ; the significance of this finding is briefly discussed.

The authors consider that the cultural results described justify a wide use of stool culture in the epidemiological investigation of similar outbreaks, and that, when full facilities exist, stool cultures of suspected carriers should not be postponed pending preliminary " serological screening ", which may yield false negatives. No epidemiological investigation can however be considered complete until supplemented by serological tests.

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

POST-INFECTIOUS ENCEPHALITIS IN 1950

(A Preliminary Report)

E. T. Conybeare, O.B.E., M.D., F.R.C.P., Ministry of Health

Cases of acute encephalitis which either accompanied or followed attacks of infectious disease such as measles, mumps, chicken pox, etc., were made notifiable from the 1st January, 1950, by the Public Health (Acute Poliomyelitis, Acute Encephalitis and Meningococcal Infections) Regulations, 1949. During 1950 a total of 71 such cases were notified and recorded under Category K in Table IV of the Registrar General's Weekly Return for England and Wales, this being a figure subject to revision as a result of the corrections* now submitted quarterly to the Registrar General. After consultation with the Society of Medical Officers of Health, local health departments have been asked to give in respect of each of these cases certain information supplementary to that available from the Registrar General. Such information was, in fact, received from medical officers of health regarding a total of 65 cases notified in 1950. A preliminary analysis of this information shows the following points which may be of interest:—

1. *Amended Diagnosis*

(a) *Polioencephalitis.* In 6 of the notified cases the disease associated with the encephalitis was subsequently judged to be poliomyelitis, and the notification was accordingly corrected.

(b) *Acute Encephalitis of Unknown Origin.* In 8 of the notified cases, although the information obtained was consistent with the diagnosis of acute encephalitis, the implied association with an infectious disease such as measles or mumps could not be substantiated. Had this been appreciated when the original notification was made these 8 cases would have been notified simply as acute encephalitis and placed accordingly in category G acute encephalitis (infective) of Table IV of the Registrar General's Weekly Return.

(c) *Not Encephalitis.* In 10 of the notified cases the diagnosis was subsequently amended to one of some disease other than acute encephalitis or poliomyelitis, and the notification was withdrawn.

2. *Post-Infectious Encephalitis*

In 41 of the notified cases it was found that the original diagnosis and inclusion in Category K of Table IV of the Registrar General's Weekly Return could be upheld. The infectious diseases associated with these 41 cases, the number of cases, and the number of deaths is shown in the following table:—

Infectious Disease	(1) Cases notified with Acute Encephalitis (Post Infectious)	(2) Fatal Encephalitis Cases included in (1)
Measles	15	4
Mumps	10	0
Chicken pox	9	1
Influenza	2	1
Tonsilitis and Pharyngitis	2	0
Vaccinia	2	0
Rubella	1	0

In view of the limited number of notified cases associated with any particular infectious disease a more detailed analysis of the information available for 1950 will not at present be attempted. It is proposed, however, to invite the continued co-operation of medical officers of health over a further period until such time as the total number of cases on which information has been received warrants a more detailed examination.

**Note.*—The corrected figures for the first three quarters of the year (50 compared with 53 original notifications) suggest that there are almost as many changes of diagnosis from other diseases to acute encephalitis (post-infectious) as there are from that disease to others. The above report takes account only of changes in the latter sense.

SOCIAL STATUS AND ACTIVE PREVENTION OF DISEASE

Israel Gordon, M.D. (Edin.), M.R.C.P. (Lond.), D.P.H.,
Medical Officer of Health, Ilford

Whilst investigating the vaccinal state of children attending the infant welfare centres of Ilford, in a week in March, 1950, some interesting facts came to light. Of 909 children, attending 15 centres, the number who had at any time been vaccinated was 308, or 34 per cent. However, the figures for each centre varied considerably, ranging from 22 per cent. to 54 per cent. A rapid survey showed immediately that the higher percentages were in the more prosperous parts of the town. The centres were divided into three groups, those serving the higher income groups (A), the less well off (B), and the poorest (C). The results were as follows :—

Totals	*Number Vaccinated*	*Percentage Vaccinated* Per cent.	*Percentage in district attending I.W.C.* Per cent.
A. 242 (3 clinics)	115	48	74
B. 404 (7 clinics)	122	30	80
C. 263 (5 clinics)	71	27	77

The difference between A and C is statistically significant.

TABLE I

The question at once arises, to what extent are these figures representative of the population of the borough as a whole, i.e., is the population of the centres a true sample? In the fourth column of table I is the percentage attendance at the infant welfare centres of the children born in that district (i.e., first visits of children under one year as a percentage of district births). It will be seen that the figure for the best districts (in vaccination) is 74 per cent., and the worst 77 per cent. The difference in attendance is so small and the difference in vaccination is so large that an error of sampling cannot account for the latter. In 1949 there were 2,630 births in the town and 726 notifications of vaccination, a percentage of 27·5. It seems reasonable to assume that the infant welfare centre figure is higher by 6·5 per cent. for the following reasons. First, some vaccinations were probably performed without being notified, and secondly, the infant welfare centre figures included a few children over one year of age, and as the vaccination rate is steadily decreasing (Conybeare 1950), a larger proportion of these older children were probably protected.

Health visitors, midwives and doctors at the clinics all advise vaccination and carry out similar propaganda. No single doctor or nurse was common to the three clinics with the best records, or to the five with the worst. There are 73 general practitioners in the town, so the differences are hardly likely to be due to any individual idiosyncrasies. Different towns, for no known reason, have extraordinarily different vaccination rates (Conybeare 1950). It would be interesting to find out if social differences exist within the range of figures of these local authorities. I fully realise that this district distribution is only an indirect approach to social status; figures for each occupational group would have been more reliable, but these figures were originally obtained for other purposes.

Vaccination is troublesome and carries no immediate advantage; the advantage lies in the future, and it is free to all sections of the population. It seemed an interesting line of enquiry to find out if there were similar social differences with respect to other prophylactic measures fulfilling similar criteria. Immunisation against diphtheria immediately comes to mind. As figures have already been prepared by the Joint Committee of the Population Committee of the Royal College of Obstetricians and Gynaecologists and the Institute of Child Health (Rowntree 1950), it was unnecessary to procure figures from Ilford, but it must be remembered that as these figures date from before the introduction of the National Health Service Act, some of the immunisations were carried out by private doctors for a fee, doubtless amongst the more well to do. The figures are:—

Professional and Salaried Workers	*Black-coated Wage Earners*	*Manual Workers*	*Agricultural Workers*	*Others*
Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
87·5	77·1	68·0	67·5	79·9

In general it appeared that immunisation was least frequent among children brought up in over-crowded homes.

Ante-natal supervision equally fulfils the criteria of lack of immediate benefit, perhaps at the cost of some discomfort, but in Ilford it is impossible to obtain statistically relevant data with respect to social status, as here ante-natal clinics only serve hospital cases, which are a selected group. The aforementioned Joint Committee have, however, compiled some data, but their figures, as for the immunisation ones, refer to a period when the wealthier parents could pay for care, and thus might well have been persuaded by their doctors. It is impossible to state how many of those attending local authority clinics were selected as in Ilford. The figures are as follows:—

(Maternity in Great Britain 1948), for the first Trimester.

Professional and Salaried Workers	*Black-coated Workers*	*Manual Workers*	*Agricultural Workers*	*Others*
Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
61·4	47·0	36·6	33·3	40

Similar qualifications must be extended to the percentage incidence of post-natal examinations, where the Joint Committee's figures for the first birth are:—

(Douglas and Rowntree 1949.)

Professional and Salaried Workers	*Black-coated Workers*	*Manual Workers*	*Agricultural Workers*
Per cent.	Per cent.	Per cent.	Per cent.
70·4	57·7	50·8	45·2

The Joint Committee also have figures for attendance at Infant Welfare Centres, but for my purpose they are of little use as the wealthier mothers who went to their private doctors are not included, and if they are, there is no evidence that they attended their private doctors for reasons that other mothers may have attended infant welfare centres, for example to buy dried milk, perhaps, or gossip and drink a cup of tea. The Ilford figures in the fourth column of Table I are equally equivocal.

A rather different line of approach is the comparative uptake of free vitamin foods, that is, cod liver oil and vital A & D tablets, distributed for infants and mothers. The Area Food Office of the London Region issues a table quarterly, and I quote some data from the 13 weeks ending 25th February 1950. Excluding the City of London, the figures for which are too small, there are 93 areas in the London Region. The dozen heading the list for Cod Liver Oil are Wood Green, Kingston, Barnet, Finsbury, Beckenham, Potters Bar, Orpington, Southgate, Bromley, Harrow, Richmond, Sutton and Cheam ; those at the bottom of the list are Poplar, Bethnal Green, Southwark, Battersea, Fulham, Greenwich, Kensington, St. Marylebone, Stepney, Islington, Shoreditch and Bermondsey. The data for vitamin tablets are very similar. It will be noticed that those boroughs at the top of the list are predominantly middle class residential boroughs largely in the North and West, those at the bottom of the list mainly working class areas in the centre, east and south.

Discussion

One of the worthy objects of our national social welfare is to provide such safeguards for every section of the population as may prevent disease, and in the examples chosen, these are all provided free. However, it seems probable that the middle classes are making more use of these facilities than the poorer classes, and we find that those who most need these safeguards are not making the use of them that they should. Thus mere free provision does not mean full utilisation.

It is interesting to speculate why the less well-off take less interest in prophylaxis. No doubt some will conclude that it is because they are less intelligent, but this is a very facile assumption and ignores other more substantial factors. Though now-a-days nearly all mothers with young children are hard worked and have little household help, yet working class mothers usually have larger families and even less help, as well as less money for fares, and so have little time or opportunity to attend clinics. A still more important possible factor is that the working class parent has not had the education available to the parent of the middle class, and it is education, above all, that is most likely to persuade the mother to make her child pay the small price in present discomfort for future security.

Conclusion

Vaccination, immunisation, ante-natal and post-natal supervision, uptake of vitamin foods are preventive measures provided free ; yet advantage of them is taken to a greater extent by the middle classes than by the poorer classes. Some of the reasons for this are discussed. It is suggested that education is as important as free provision.

I would like to thank the Superintendent of Health Visitors and the health visitors for their help in collecting data relevant to the infant welfare centres.

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DAY NURSERY DIETARIES

H. G. Cairney, M.B.E., Dietitian, Ministry of Health.

Correct feeding in childhood is of the greatest importance because only by giving a well balanced diet, that is one containing all the essential nutrients, can the child grow normally and attain maximal physical development. A satisfactory weight does not necessarily show that the child is having all the essential food stuffs.

Three years ago it was suggested that it would help matrons of day nurseries if they could have expert guidance on their catering problems and it was therefore arranged that a dietitian should visit as many nurseries as possible and give them the necessary help. 350-400 nurseries have now been visited and several salient facts have emerged. The dietaries have been found to vary much and although advances have been made there is still room for improvement.

To give well balanced meals one must first understand thoroughly the rationing system, and, secondly, make the best possible use of the rations allowed.

Rations for day nurseries are similar to those of the " school meals " ration system. These allowances are based on the number of meals returned on Form E.G.C.3 every 16 weeks to the local Food Office and not, as has been often thought, on the number of places for children and staff. It is, therefore, most important that every meal should be recorded. Ration allowances are set out on Page 11 of " Our food to-day No. 3 " which is a Ministry of Food publication obtainable from the Public Relations Department, Dean Bradley House, Horseferry Road, S.W.1. This Ministry also issues a supplement to this book every 8 weeks giving the current ration allowances and these must be obtained to keep up to date with the changing rations. With this information it is easy to work out the amount of rationed foods to which a particular nursery is entitled and to check these with the permits in order to see that the full allowances are being obtained. These allowances should be checked periodically against the record of meals served and, if necessary, permits revised by application to the local Food Office.

It is important that the full rations of meat, bacon, cheese and butter be bought though it may not be necessary to take up the entire allowance of margarine and sugar. Milk is allowed at the rate of 2/3rds pint for each child in attendance at the nursery for the whole day and in addition 20 pints per 100 main meals and hot beverages served ; both these milk allowances should be fully drawn.

Having taken up the rations the next step to a well balanced diet is to use them correctly. Many nurseries now open later and, dispensing with the cooked breakfast, give milk and cereal or rusks, but in nurseries which open early it is advisable to give a more substantial meal. Indeed, it is important that the matron should know from the mothers of the arrangements they make at home for breakfast and tea so that these meals may be suitably provided at the nursery for each individual child. Dinners are on the whole fairly good but there is still a tendency to give too little meat. Too often the meat is minced and served with too much gravy, too little green or root vegetables and too much potato. If the full meat ration is taken it will provide the equivalent of one table-spoonful for each child under 2 years of age and 1½-2 tablespoonfuls for each toddler for all dinners except one each week, when fish or offal should be given. When purchasing fish for a main meal, a useful guide is to allow 1 lb. of filleted fish to 4-5 children. Liver is not plentiful but it should be possible to obtain sufficient to give each child a dinner at least once a month, the quantity required is 1 lb. liver to 5-6 children. One medium sized potato for each child should be sufficient and more variety in the cooking of these could and should be introduced, when they are mashed the Vitamin C content is considerably reduced. When buying green vegetables 1 lb. to 4-5 children should be allowed. Neither potatoes nor other vegetables should be soaked for long before cooking as in this way valuable mineral salts and vitamins are lost. There is a lack of variety in puddings served and the milk pudding still predominates in some nurseries.

Some hold the view that the nursery child, having had a good dinner at 12 mid-day, will require only bread and jam, cakes or biscuits for tea at 3.30-4 p.m. This is wrong because after an active time out of doors the child will be hungry and with a little encouragement will enjoy a substantial tea. If this consists entirely of starchy foods the balance of the day's meals is upset. By giving a protein food at this meal less starchy foods are eaten and it will be found that over a period the cost of providing a protein for tea varies little from the cost of providing large quantities of carbo-hydrate. For instance, if the full cheese ration is bought it will allow of ¾ oz. for each child at a meal for 3 teas in 2 weeks at a cost of ¾d. per meal. The bacon ration of 1 oz.-2 oz. for each child at a meal will provide 4-5 teas of bacon or ham in two weeks at a cost of 2d.-3d. per meal. Herrings, which are an excellent source of vitamins A and D can be given to toddlers at a cost of 1½d.-2d. per meal, while the under 2 year olds can have roes. Milk jellies, junkets or moulds made from the catering and not the drinking milk allowance can be given at a cost of 1½d. per child. These are good protein foods and there are many others which can be used. If one of these is served at the tea meal each day with a salad, when available, and ⅓rd pint of milk with bread and butter the child is given a well balanced meal. It is not a good plan to put the protein in sandwiches because this gives no guide as to the amount of protein consumed by the individual child. It is appreciated that while some children may be given a cooked supper many only have bread and jam on their return home. The cooked meal is unlikely to contain much in the way of first class protein and to give it just before bedtime is not in any case nutritionally sound. If a good tea is given in the nursery it should not be necessary to give more than a milky drink and biscuit at bedtime.

The importance of giving each child his full ⅔rds pint of drinking milk each day cannot be over-stressed. Most of the beakers used in the nurseries are not large enough to hold ⅓rd pint comfortably. They are often only half filled to begin with and may not be replenished. One way in which the matron can ensure that each child has his full allowance is to see that the person in charge of the group measures into a jug the number of ⅓rds of a pint required and systematically refills each beaker until the jug is empty.

For the child who does not like milk there are a number of ways of making it attractive. Children love colour and, with the reliable flavourings and colourings now available, it is possible to make milk drinks which will appeal to the children. Every meal should be planned to introduce colour and should be correctly cooked and attractively served.

A dietary containing the essential foods has been worked out at a cost of 1s. 2½d. per child per day, a surprisingly small proportion of the total cost of maintenance in the Day Nursery.

The following summarises such a diet:—

Light Breakfast—8.30-9 a.m.

⅓rd pint milk.
Porridge or Cereal and rusks, butter and marmite.

Dinner—12 mid-day.
- Meat or fish.
- One Potato.
- Greens, root vegetable, or salad.
- Pudding, steamed or baked or milk pudding and fruit.
- Raw apple or carrot.
- Water to drink.

Tea—4 p.m.
- Cheese, fish, bacon or ham.
- Salad vegetables as in season including raw cabbage.
- Brown bread and butter.
- ⅓rd pint of milk.
- Either jam, honey, treacle or home made biscuits.

There is much helpful information in the booklet published by the Ministry of Health, " Feeding the One to Fives " (H.M. Stationery Office ; 6d.), and it should be in the hands of every nursery matron.

NOTIFICATION OF INFECTIOUS DISEASES IN ENGLAND AND WALES, JANUARY, 1951

Issued from the General Register Office, Somerset House, W.C.2

	January 6th	January 13th	January 20th	January 27th	Average weekly figures for January, 1950
Scarlet Fever	923	853	1,015	956	1,887
Whooping Cough	5,011	4,980	4,822	5,025	1,608
Diphtheria	38	30	43	48	80
Measles, excluding Rubella ...	21,691	18,658	15,996	15,942	2,382
Acute Pneumonia	1,774	2,538	3,112	3,572	977
Meningococcal Infection ...	50	61	44	60	40
Acute Poliomyelitis (Paralytic)...	37	27	29	20	40
Acute Poliomyelitis (Non-paralytic)	10	11	7	7	14
Ophthalmia Neonatorum ...	26	19	31	11	42
Puerperal Pyrexia and Puerperal Sepsis	90	102	107	102	87
Dysentery	834	698	727	706	170
Paratyphoid	1	8	2	4	3
Typhoid	4	—	4	4	12
Smallpox	13	11	3	2	—

No cases of Cholera or Plague or Tpyhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Hereford Laboratory : change of address

The laboratory has moved to new quarters at the following address: County Hospital, Hereford (*Tel. :* Hereford 4696).

Toxoplasmosis

Dr. I. A. B. Cathie and Dr. J. A. Dudgeon of the Department of Clinical Pathology, Hospital for Sick Children, Great Ormond Street, London, W.C.1 (*Tel. :* Holborn 9200, Ext. 24) have kindly agreed to act as reference experts for the examination of material from suspected cases of toxoplasmosis.

Five ml. of fresh serum, unheated and without addition of any antiseptic, should be submitted for examination. When the congenital form of the disease is suspected, serum should be taken from the mother as well as from the patient. Attempts to isolate the causative organism are best made locally, but post-mortem material may be sent for histological examination.

To avoid possible delay, specimens should be addressed to the Clinical Pathologist rather than to Dr. Cathie or Dr. Dudgeon personally.

Contagious Pustular Dermatitis

A few cases of local infection with this disease have been recognized among butchers in Liverpool. The lesion in man bears some resemblance to a primary vaccination take, and may be found on the hands or arms of persons handling sheep before or after slaughter. Dr. A. Macdonald would be grateful for the following material from human cases: (*a*) vesicle or pustule fluid in capillary tubes or a piece of the eschar; (*b*) serum taken three or more weeks after the onset of the disease. It should be addressed to him at the University Department of Bacteriology, Liverpool, 3.

Hyperglobulinaemia

Dr. J. Marks would be glad to receive sera containing excess globulin from patients suffering from *non-infective* diseases for the purpose of studying normal antibody production. Specimens (not less than 1 ml.) should be sent to the Public Health Laboratory, The Parade, Cardiff, with the patient's name, age, sex, diagnosis and serum protein values.

Dysentery Reference Laboratory

Dr. Patricia Carpenter would be grateful for strains of *Shigella* or shigella-like organisms isolated from animals of any kind. A short history should accompany the culture. Cultures should be addressed to Dr. Carpenter at the Dysentery Reference Laboratory, Public Health Laboratory, Walton Street, Oxford.

Correction

October, 1950, *number, p.* 235 (*Table III*).—The number of Grade 2 samples examined during 1947 in which presumptive coliform organisms were present in 1/10 ml. after pre-incubation is given as 60 (22.6 per cent.). The figure should have been 160 (60.4 per cent.).

PUBLIC HEALTH LABORATORY SERVICE

Medical Research Council
(*March*, 1951)

ADMINISTRATIVE HEADQUARTERS:
38, Old Queen Street, Westminster, London, S.W.1
Tel.: Whitehall 4884

Chairman of the Public Health Laboratory Service Board: Dr. A. Landsborough Thomson.

Director of the Service: Dr. G. S. Wilson.

CENTRAL PUBLIC HEALTH LABORATORY:
Colindale Avenue, London, N.W.9.
Tel.: Colindale 6041/2 and 4081/2.

Director: Lt.-Col. H. J. Bensted.

A.—CONSTITUENT LABORATORIES

England

Place	*Address*	*Pathologist in charge*	*Telephone number*
Bedford	Public Health Laboratory, St. Peter's Hospital. (To be opened shortly.)		
Birkenhead	Public Health Laboratory, 42, Hamilton Square, Birkenhead.	Dr. J. Morrison Ritchie.	Birkenhead 5484
Birmingham	Public Health Laboratory, 150, Great Charles St., Birmingham 3.	Dr. B. R. Sandiford.	Central 1724
Bradford	Public Health Laboratory, 16–18, Edmund Street, Bradford.	Dr. A. J. H. Tomlinson.	Bradford 24314
Brighton	Public Health Laboratory, Royal Sussex County Hospital, Brighton 7.	Dr. J. E. Jameson.	Brighton 23506
Cambridge	Public Health Laboratory, Tennis Court Road, Cambridge.	Dr. R. M. Fry	Cambridge 55526
Coventry	Public Health Laboratory, Stoney Stanton Road, Coventry.	Dr. R. E. Jones	Coventry 63256–7 and 5937
Dorchester	Public Health Laboratory, Glyde Path Road, Dorchester, Dorset. (To be opened shortly.)		
Epsom	Public Health Laboratory, West Hill House, West Hill, Epsom, Surrey.	Dr. D. M. Stone	Epsom 2474
Exeter	Public Health Laboratory, 7, Dix's Field, Exeter.	Dr. B. Moore	Exeter 54959
Harrogate	Public Health Laboratory, Harrogate and District General Hospital. (To be opened shortly.)		
Hereford	Public Health Laboratory, County Hospital, Hereford.	Dr. D. R. Christie	Hereford 4696
Hull	Public Health Laboratory, 184, High St., Kingston-upon-Hull.	Dr. C. L. Greening	Hull 35371
Ipswich	Public Health Laboratory, Borough General Hospital, Woodbridge Road, Ipswich.	Dr. P. H. Martin	Ipswich 77261/2
Leicester	Public Health Laboratory, Isolation Hospital, Groby Road, Leicester.	Dr. E. H. Gillespie.	Anstey 2383

England—*continued*

Place	*Address*	*Pathologist in charge*	*Telephone number*
Lincoln	Public Health Laboratory, St. Anne's Road, Lincoln.	Dr. J. M. Croll	Lincoln 8607
London (Camberwell).	Public Health Laboratory, 134, Denmark Hill, London, S.E.5.	Dr. G. Bousfield	Brixton 1188
London (Colindale).	Central Public Health Laboratory, Colindale Avenue, London, N.W.9.	Lt.-Col. H. J. Bensted.	Colindale 6041 and 4081
London (Hither Green).	Public Health Laboratory, Park Hospital, London, S.E.13.	Dr. J. E. McCartney.	Hither Green 4417
Luton	Public Health Laboratory, Town Hall, Luton.	Dr. J. H. C. Walker.	Luton 2800
Manchester	Public Health Laboratory, Monsall Hospital, Newton Heath, Manchester 10.	Dr. M. T. Parker	Collyhurst 2733
Middlesbrough	Public Health Laboratory, General Hospital, Ayresome Green Lane, Middlesbrough. (To be opened shortly.)		
Newcastle-on-Tyne.	(*a*) Public Health Laboratory, General Hospital, Westgate Road, Newcastle-on-Tyne 4.	Dr. A. I. Messer	Newcastle 34920
	(*b*) Public Health Laboratory, Government Buildings, Ponteland Road, Newcastle-on-Tyne 5.	Dr. R. Norton	Kenton 69319
Northallerton	Public Health Laboratory, County Hall, Northallerton, Yorks.	Dr. D. J. H. Payne (acting).	Northallerton 88
Northampton	Public Health Laboratory, General Hospital, Northampton.	Dr. L. Hoyle	Northampton 347
Norwich	Public Health Laboratory, Isolation Hospital, Bowthorpe Road, Norwich.	Dr. L. M. Dowsett.	Norwich 21095
Nottingham	Public Health Laboratory, 63, Goldsmith Street, Nottingham.	Dr. G. B. Ludlam	Nottingham 41304
Oxford	Public Health Laboratory, Walton Street, Oxford.	Dr. R. L. Vollum	Oxford 47884/5
Peterborough	Public Health Laboratory, Peterborough and District Memorial Hospital, Midland Road, Peterborough.	Dr. C. C. B. Gilmour.	Peterborough 2277
Poole	Public Health Laboratory, Municipal Buildings, Poole, Dorset.	Dr. G. J. G. King	Poole 393, Ext. 13
Portsmouth	Public Health Laboratory, "G" Block, Infectious Diseases Hospital, Portsmouth.	Dr. K. E. Hughes	Portsmouth 74785/6/7
Reading	Public Health Laboratory, Battle Hospital, Reading. (From 1st April.)	Dr. N. Wood	Reading
Sheffield	Public Health Laboratory, City General Hospital, Sheffield 5.	Dr. L. G. Cook (acting.)	Sheffield 36751
Shrewsbury	Public Health Laboratory, Royal Salop Infirmary, Shrewsbury.	Dr. A. C. Jones	Shrewsbury 4389
Southampton	Public Health Laboratory, The Health Centre, King's Park Road, Southampton.	Dr. R. I. Hutchinson.	Southampton 3788
Southend	Public Health Laboratory, Westcliff Hospital, Balmoral Road, Southend-on-Sea.	Dr. R. Pilsworth	Southend 45440
Stafford	Public Health Laboratory, Martin Street, Stafford.	Dr. R. N. Phease	Stafford 377
Sunderland	Public Health Laboratory, Royal Infirmary, Sunderland.	Dr. P. B. Crone	Sunderland 5492
Taunton	Public Health Laboratory, County Hall, Taunton.	Dr. J. A. Boycott	Taunton 3451

England—*continued*

Place	*Address*	*Pathologist in charge*	*Telephone number*
Truro	Public Health Laboratory, Royal Cornwall Infirmary, Truro.	Dr. F. D. M. Hocking. (acting).	Truro 3029
Wakefield ...	Public Health Laboratory, County Medical Offices, Wood Street, Wakefield, Yorks.	Dr. W. F. Lane...	Wakefield 2207
Watford ...	Public Health Laboratory, Peace Memorial Hospital, Watford.	Dr. B. H. E. Cadness-Graves.	Watford 2369
Winchester ...	Public Health Laboratory, Royal Hants County Hospital, Winchester.	Dr. R. D. Mackenzie.	Winchester 3807
Worcester ...	Public Health Laboratory, Royal Infirmary, Worcester.	Dr. R. J. Henderson.	Worcester 2697

Wales

Aberystwyth ...	Public Health Laboratory, Cardiganshire General Hospital, Aberystwyth.	Dr. M. V. N. Sudds.	Aberystwyth 216
Cardiff	Public Health Laboratory, Institute of Preventive Medicine, The Parade, Cardiff.	Dr. Scott Thomson.	Cardiff 8288 and 5827
Carmarthen ...	Public Health Laboratory, Penlan Road, Carmarthen.	Dr. W. Kwantes (acting.)	Carmarthen 7271
Conway ...	Public Health Laboratory, "Bryn Hyfryd," Conway.	Dr. A. J. Kingsley Smith.	Conway 2178
Newport (Mon.)	Public Health Laboratory, County Hall, Newport, Mon.	Dr. R. D. Gray...	Newport 4955

B.—ASSOCIATED LABORATORIES

England

Bristol	Department of Preventive Medicine, Whatley Road, Clifton, Bristol 8.	Prof. K. E. Cooper	Bristol 38257
Carlisle ...	Cumberland Pathological Laboratory, Cumberland Infirmary, Carlisle.	Dr. J. S. Faulds...	Carlisle 590
Derby	County Bacteriological Laboratory, County Offices, St. Mary's Gate, Derby.	Dr. J. L. G. Iredale.	Derby 47131, Ext. 120
*Dorchester ...	County Laboratory, Glyde Path Road, Dorchester, Dorset.	Dr. T. V. Cooper	Dorchester 600
Leeds	Bacteriological Department, School of Medicine, Leeds.	Prof. J. W. McLeod.	Leeds 20071
Liverpool ...	Bacteriology Department, City Laboratories, 126, Mount Pleasant, Liverpool 3.	Prof. D. T. Robinson.	Royal 3636/7
Maidstone ...	County Laboratory, County Hall, Maidstone.	Dr. E. R. Jones...	Maidstone 4321, Ext.286
Plymouth ...	Pathological Department, South Devon and East Cornwall Hospital, Greenbank Rd., Plymouth.	Dr. E. Wordley...	Plymouth 2737
Salisbury ...	Pathological Department, General Infirmary, Salisbury.	Dr. L. H. D. Thornton.	Salisbury 2950

Wales

Swansea ...	Pathological Department, Beck Laboratory, General Hospital, Swansea.	Dr. A. F. S. Sladden.	Swansea 2001

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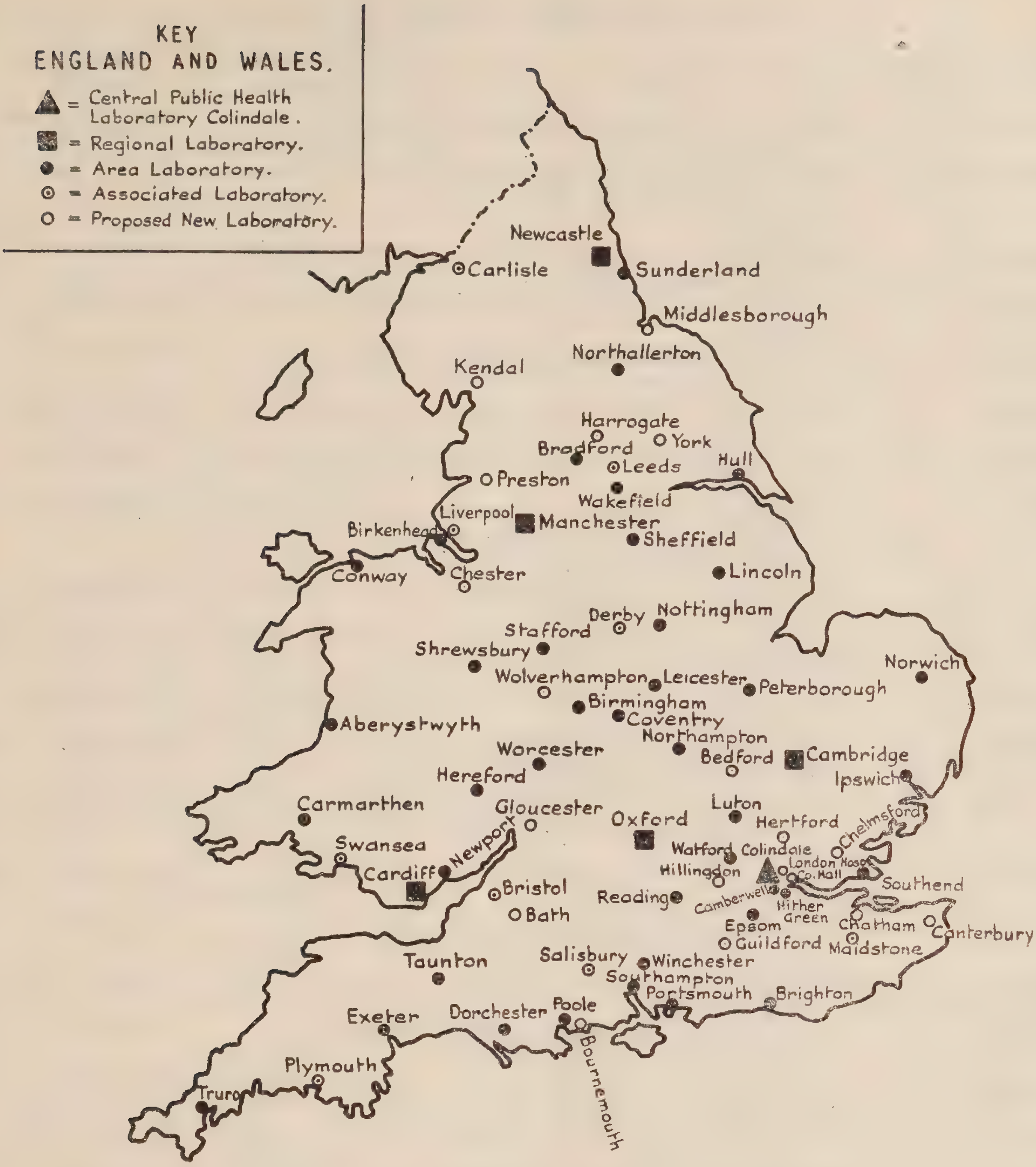
* To be replaced shortly by a constituent laboratory (see p. 66).



Map of England and Wales showing present and proposed future distribution of public health laboratories (Recognized laboratories are not included).

C.—RECOGNIZED LABORATORIES

Place	*Address*	*Pathologist in charge*	*Telephone number*
Aylesbury	Ministry of Pensions Hospital, Stoke Mandeville.	Dr. C. L. Greenbury.	Aylesbury 900
Barrow	Group Pathological Laboratory, 64, School Street, Barrow-in-Furness.	Dr. J. E. Horrocks	Barrow 1098
Blackburn	Pathological Laboratory, Blackburn Royal Infirmary.	Dr. M. S. Spink	Blackburn 7133
Blackpool	Pathological Laboratory, Victoria Hospital.	Dr. F. H. Yates	Blackpool 23881
Bolton	Pathological Laboratory, Bolton Royal Infirmary.	Dr. G. B. Manning.	Bolton 5250
Burnley	Pathological Laboratory, Victoria Hospital.	Dr. M. Behr	Burnley 3237
Chester	Pathological Laboratory, Royal Infirmary.	Dr. W. H. Grace	Chester 2842
Ealing	Pathological Laboratory, King Edward Memorial Hospital, Mattock Lane, W.13.	Dr. W. H. Hughes	Ealing 4081
Eastbourne	Department of Pathology, Princess Alice Memorial Hospital.	Dr. A. G. Shera	Eastbourne 3444
Gloucester	Department of Pathology, Royal Infirmary.	Dr. E. N. Davey	Gloucester 3584
Hastings	Department of Pathology, Royal East Sussex Hospital.	Dr. P. Lazarus-Barlow.	Hastings 285
Hellingly	Department of Pathology, Hellingly Hospital, Hailsham, Sussex.	Dr. A. G. Shera	Hellingly 318/9
Kettering	Department of Pathology, Kettering and District General Hospital.	Dr. H. G. Voss	Kettering 2841
Lancaster	Pathological Laboratory, Royal Lancaster Infirmary.	Dr. A. G. Rickards.	Lancaster 1200
Oldham	Pathological Laboratory, Oldham Royal Infirmary.	Dr. C. H. Adderley.	Main 3828
Preston	Pathological Laboratory, Preston Royal Infirmary.	Dr. A. A. Miller	Preston 4616
Salford	Pathological Laboratory, Hope Hospital, Salford 6, Lancs.	Dr. G. J. Crawford.	Eccles 1511
Stockport	Pathological Laboratory, Stepping Hill Hospital.	Dr. J. C. Kerrin	Gt. Moor 3821/4
Wigan	Pathological Laboratory, Wigan Royal Infirmary.	Dr. J. L. Dales	Wigan 2841/3
York	Pathological Laboratory, County Hospital.	Dr. S. G. Platts	York 3014

D.—REFERENCE LABORATORIES

Central Enteric Reference Laboratory and Bureau:
Director: Dr. A. Felix.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Dysentery Reference Laboratory:
Consulting Director: Lt.-Col. R. F. Bridges.
Assistant Director: Dr. Patricia Carpenter.
Address: Public Health Laboratory, Walton Street, Oxford. *Tel.:* Oxford 47884/5.

Malaria Reference Laboratory:
Director: Sir Gordon Covell.
Address: Horton Hospital, Epsom. *Tel.:* Epsom 2343.

Mycological Reference Laboratory:
Acting Director: Dr. Jacqueline Walker.
Address: London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Salmonella Reference Laboratory:
Director: Dr. Joan Taylor.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Streptococcus and Staphylococcus Reference Laboratories:
Director: Dr. R. E. O. Williams.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Venereal Diseases Reference Laboratory:
Director: Dr. I. N. Orpwood Price.
Address: St. Peter's Hospital, Vallance Road, Whitechapel, London, E.1. *Tel.:* Bishopsgate 5896.

Virus Reference Laboratory:
Director: Dr. F. O. MacCallum.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

E.—SPECIAL LABORATORIES

Air Hygiene Unit:
Director: Dr. R. E. O. Williams.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Epidemiological Research Unit:
Director: Dr. W. Charles Cockburn.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Food Hygiene Unit:
Director: Dr. Betty C. Hobbs.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

National Collection of Type Cultures:
Director: Dr. S. T. Cowan.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Standards Laboratory for Serological Reagents:
Director: Lt.-Col. H. J. Bensted.
Address: Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

F.—REFERENCE EXPERTS

(*For the examination of materials other than those normally submitted to the Reference laboratories in List D.*)

Dr. C. H. Andrewes (Influenza):
National Institute for Medical Research, The Ridgeway, Mill Hill, London, N.W.7. *Tel.:* Mill Hill 3666.

Prof. S. P. Bedson (Psittacosis):
London Hospital, Whitechapel, London, E.1. *Tel.:* Bishopsgate 3255.

Lt.-Col. H. J. Bensted (Meningococci):
Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Dr. J. C. Broom (Leptospiral infections):
Wellcome Laboratories for Tropical Medicine, 183, Euston Road, London, N.W.1. *Tel.:* Euston 4688.

Prof. J. J. C. Buckley (Helminthology):
London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Prof. P. A. Buxton (Entomology):
London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Dr. I. A. B. Cathie (Toxoplasmosis):
Department of Clinical Pathology, Hospital for Sick Children, Great Ormond Street, London, W.C.1. *Tel.:* Holborn 9200, Ext. 24.

Prof. J. C. Cruickshank (Anaerobes and Brucella):
London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Prof. A. W. Downie (Smallpox):
Bacteriology Department, School of Hygiene, Mount Pleasant, Liverpool, 3. *Tel.:* Royal 6301.

Dr. J. A. Dudgeon (Toxoplasmosis):
Department of Clinical Pathology, Hospital for Sick Children, Great Ormond Street, London, W.C.1. *Tel.:* Holborn 9200, Ext. 24.

Dr. A. Felix (Typhus fever):
Central Enteric Reference Laboratory, Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Dr. R. M. Fry (Typing of Group A streptococci):
Public Health Laboratory, Tennis Court Road, Cambridge. *Tel.:* Cambridge 55526.

Dr. C. C. B. Gilmour (Cholera vibrios):
Public Health Laboratory, Peterborough and District Memorial Hospital, Midland Road, Peterborough. *Tel.:* Peterborough 2277.

Dr. Betty C. Hobbs (Food poisoning):
Food Hygiene Unit, Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Dr. L. Hoyle (Influenza):
Public Health Laboratory, General Hospital, Northampton. *Tel.:* Northampton 347.

Dr. M. T. Parker (Plague):
Public Health Laboratory, Monsall Hospital, Manchester, 10. *Tel.:* Collyhurst 2733.

Prof. D. T. Robinson (Diphtheria):
Bacteriological Department, City Laboratories, 126, Mount Pleasant, Liverpool, 3. *Tel.:* Royal 3636/7.

Prof. H. E. Shortt (Protozoology):
London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Dr. Joan Taylor (Typing of strains of *Bact.coli* associated with infantile enteritis):
Salmonella Reference Laboratory, Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

Dr. Scott Thomson (Typing of Group A streptococci):
Public Health Laboratory, The Parade, Cardiff. *Tel.:* Cardiff 8288 and 5827.

Dr. A. J. H. Tomlinson (Anthrax):
Public Health Laboratory, 16–18, Edmund Street, Bradford. *Tel.:* Bradford 24314.

Dr. R. L. Vollum (Typing of Group A streptococci):
Public Health Laboratory, Walton Street, Oxford. *Tel.:* Oxford 47884/5.

Dr. A. Q. Wells (Tubercle bacilli):
Sir William Dunn School of Pathology, South Parks Road, Oxford. *Tel.:* Oxford 47626.

Dr. R. E. O. Williams (Pneumococci):
Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 6041/2 and 4081/2.

G.—LIST OF SPECIALIST INVESTIGATIONS AVAILABLE TO ALL LABORATORIES OPERATING UNDER THE NATIONAL HEALTH SERVICE ACT, AND TO UNIVERSITY LABORATORIES

(*Note: The letter* (*A*), (*B*), (*C*) *etc. after an expert's name is a reference to the list, given above, in which his address will be found.*)

Anaerobes, identification	Prof. J. C. Cruickshank (F).
Anthrax bacilli, identification	Dr. A. J. H. Tomlinson (F).
Brucella infections:	
(*a*) cultural tests	Prof. J. C. Cruickshank (F).
(*b*) serological tests	
Cholera vibrios, identification	Dr. C. C. B. Gilmour (F).
Diphtheria bacilli, serological typing	Prof. D. T. Robinson (F).
Dysentery bacilli, typing	Dr. Patricia Carpenter (D).
Encephalitis, acute infectious	Dr. F. O. MacCallum (D).
Enteric Fever:	
(*a*) Vi-agglutination in suspected carriers	Dr. A. Felix (D).
(*b*) Vi-phage-type determination of strains of typhoid and paratyphoid bacilli	
(*c*) Vi-phage-type determination of strains of *Salmonella typhi-murium* (from outbreaks only)	
Enteritis, infantile, typing of strains of *Bact.coli*	Dr. Joan Taylor (F).

Entomological specimens, investigation	Prof. P. A. Buxton (F).
Food Poisoning	Dr. Betty Hobbs (E).

Note: Owing to the perishable nature of most foodstuffs, material for investigation from outbreaks of food poisoning should normally be sent to the nearest public health laboratory. The reference laboratory should be used mainly for non-perishable articles of food, especially when litigation may arise, and for the identification of strains.

Fungi (pathogenic), identification	Dr. Jacqueline Walker (D).
Helminthological specimens, investigation	Prof. J. J. C. Buckley (F).
Influenza:	
(*a*) Cultural tests	Dr. C. H. Andrewes (F). Dr. F. O. MacCallum (D).
(From the area within a 30-mile radius of Northampton only)	Dr. L. Hoyle (F).
(*b*) serological tests:	
(i) country north of a line joining the Wash and the mouth of the River Severn	Dr. L. Hoyle (F).
(ii) country south of this line	Dr. F. O. MacCallum (D).
**Leptospiral infections*, agglutination tests	Dr. J. C. Broom (F).
Lymphocytic meningitis	Dr. F. O. MacCallum (D).
Lymphogranuloma venereum	Dr. F. O. MacCallum (D).
Malaria	Sir Gordon Covell (D).
Meningococci, typing	Lt.-Col. H. J. Bensted (F).
Plague, investigation	Dr. M. T. Parker (F).
Pneumococci, typing of from epidemics	Dr. R. E. O. Williams (F).
Pneumonia, atypical	Dr. F. O. MacCallum (D).
Poliomyelitis	Dr. F. O. MacCallum (D).
Protozoological specimens, investigation	Prof. H. E. Shortt (F).
Psittacosis, diagnosis	Prof. S. P. Bedson (F).
Q Fever	Dr. F. O. MacCallum (D).
Salmonella organisms, typing	Dr. Joan Taylor (D).
Smallpox, laboratory tests for diagnosis	Prof. A. W. Downie (F). Dr. F. O. MacCallum (D).
Staphylococci, bacteriophage typing	Dr. R. E. O. Williams (D).
Streptococci of Group A, typing:	
Streptococcus Reference Laboratory	Dr. R. E. O. Williams (D).
Regional typing laboratories:	
(i) *North:* Northumberland, Cumberland, Westmorland, Co. Durham, Lancs., Yorks. *South East:* London, Middlesex, Kent, Surrey, Sussex, Hants., Dorset	Dr. R. E. O. Williams (D).
(ii) *East:* Essex, Herts., Beds., Hunts., Cambs., Norfolk, Suffolk, Northants., Rutland, Leics., Derby., Notts., Lincs.	Dr. R. M. Fry (F).
(iii) *West:* Berks., Bucks., Oxon., Warwick, Staffs., Cheshire, Salop, Worcs., Hereford, Glos., Wilts., Somerset, Devon, Cornwall	Dr. R. L. Vollum (F).
(iv) *Wales*	Dr. Scott Thomson (F).
Toxoplasmosis	Dr. I. A. B. Cathie (F) and Dr. J. A. Dudgeon (F) (See " Toxoplasmosis " on page 65).
Trichinosis, examination of rats or pigs	Prof. J. J. C. Buckley (F).
Tubercle bacilli, typing	Dr. A. Q. Wells (F).
Typhus Fever, serological tests	Dr. A. Felix (F).

Note: The Weil-Felix test can be carried out in all constituent laboratories of the Service, and also in a number of hospital laboratories. Only sera giving a doubtful reaction should be sent to Dr. Felix.

Venereal Diseases	Dr. I. N. Orpwood Price (D).
Viruses, material from infections other than those mentioned	Dr. F. O. MacCallum (D).

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* It is hoped that, in a few months' time, three or four regional laboratories will be able to undertake preliminary examination of material from different parts of the country. A note will be published in the Monthly Bulletin when the arrangements have been completed.

H.—VACCINES AND SERA OBTAINABLE THROUGH THE PUBLIC HEALTH LABORATORY SERVICE

(*These reagents are issued free of charge to medical officers of health and to general practitioners taking part in Local Health Authorities' arrangements made under Section* 26 *of the National Health Service Act*)

Laboratory (For address see list A, B or C above)	Vaccine Lymph	Typhus Vaccine	Adult (2) (3) Measles Serum	Rabies Vaccine	Anthrax (4, 5) Antiserum
Birmingham (A) ...	+	+	+	—	—
Bradford (A) ...	—	—	—	—	+
Bristol (B)	+	+	+	—	—
Cambridge (A) ...	+	+	+	—	—
Carlisle (B) ...	+	—	—	—	—
Derby (B)	+	—	—	—	—
Dorchester (B) ...	+	—	—	—	—
Exeter (A)	+	+	+	—	—
Hereford (A) ...	+	—	—	—	—
Hull (A)	—	—	—	—	+
Ipswich (A) ...	+	—	—	—	—
Leeds (B)	+	+	+	—	—
Leicester (A) ...	+	—	—	—	—
Lincoln (A) ...	+	—	—	—	—
Liverpool (B) ...	+	+	+	+	+
London (Colindale) (A)	+	+	+	+	+
Maidstone (B) ...	+	—	—	—	—
Manchester (A) ...	+	+	+	—	—
Newcastle (*a*) (A) ...	+	+	+	+	+
Northallerton (A) ...	+	—	—	—	—
Northampton (A) ...	+	—	—	—	—
Norwich (A) ...	+	—	—	—	—
Oxford (A)	+	+	+	—	—
Preston (C)	+	—	—	—	—
Sheffield (A) ...	+	+	+	—	—
Taunton (A) ...	—	—	—	—	+
Wakefield (A) ...	+	—	—	—	—
Winchester (A) ...	+	—	—	—	—
Cardiff (A)	+	+	+	+	+
Carmarthen (A) ...	+	—	—	—	—
Conway (A)... ...	+	—	—	—	—

+ means that a stock is normally held. — means that a stock is not normally held.

NOTE 1.—*Diphtheria prophylactics* may be obtained through any Constituent or Associated laboratory of the Service.

„ 2.—*Adult measles serum* is obtainable on request from any Constituent or Associated laboratory; a fairly large stock, however, is maintained at the laboratories mentioned here.

„ 3.—The Central Public Health Laboratory at Colindale have a small quantity of *gamma globulin* for issue under strictly defined conditions.

„ 4.—For prophylactic use only.

„ 5.—The following substances for *therapeutic* use are obtainable through the Hospital Pathological Service: Anthrax antiserum, Botulinum antiserum and Snake venom antiserum. A list of the hospitals supplying these agents was published in the Monthly Bulletin, 1950, January number, p. 21.

„ 6.—A list of centres where inoculation against *yellow fever* can be carried out is obtainable from the Ministry of Health, Whitehall, London, S.W.1.

„ 7.—*T.A.B.C.*, *cholera* and most other vaccines are available commercially.

„ 8.—*Material for intradermal diagnostic tests:* Frei antigen for Lymphogranuloma inguinale, Brucellin for undulant fever, Trichina antigen for Trichinosis and Hydatid antigen for Hydatid disease can be obtained from Lt.-Col. H. J. Bensted, Standards Laboratory for Serological Reagents, Central Public Health Laboratory, Colindale Avenue, London, N.W.9. (*Tel.:* Colindale 6041/2 and 4081/2).

„ 9.—Certain reagents for the diagnosis of venereal disease can be obtained from Dr. I. N. Orpwood Price at the Venereal Diseases Reference Laboratory, St. Peter's Hospital, Vallance Road, Whitechapel, London, E.1. (*Tel.:* Bishopsgate 5896).

HUMAN INFECTION DUE TO A NEW *SALMONELLA*— *SALMONELLA SALFORD*

S. Hilda Douglas, M.Sc., and Joan Taylor, B.Sc., M.B., B.S.,
Salmonella Reference Laboratory, Colindale,

E. Greenwood, M.R.C.S., L.R.C.P., Bolton Royal Infirmary,

and

S. Ibbotson, F.I.M.L.T., Hope Hospital, Salford.

A male baby aged four months was admitted to hospital with a history of diarrhoea. Two days previously it had been vaccinated ; the same evening it passed a motion containing blood and mucus, and vomited after its feed. The following day it vomited on one occasion, and passed seven offensive motions each of which contained blood and mucus. When the stool was passed the baby drew up its legs as if in pain ; it was very restless that night. The following day a doctor was consulted, who referred the case to hospital. On admission the baby's condition was good and the temperature normal. A few relaxed green stools were passed but no blood or mucus was seen. The baby made an uninterrupted recovery and was discharged five days later. No other members of the family were affected.

Investigation

A specimen of faeces was examined and a non-lactose-fermenting organism isolated. This Gram-negative motile bacillus fermented, with the production of acid and gas, glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, dextrin, rhamnose, xylose, trehalose and inositol. It failed to ferment sucrose, salicin, adonitol, inulin or raffinose. The citrate-utilization test was positive ; hydrogen sulphide was produced, but indole was not formed ; gelatin was not liquefied and urease was not produced. The Voges-Proskauer reaction was negative, the methyl red reaction positive.

When examined serologically, this organism was agglutinated to titre by *Salm.hvittingfoss* 0 serum. Absorption of this serum by the organism completely removed the homologous agglutinins ; similarly *Salm.hvittingfoss* removed all the homologous agglutinins from an antiserum prepared with the organism. Therefore the somatic antigen is XVI.

The culture was plated and single colonies tested ; some were agglutinated by *Salm.london* H (l.v) serum and some by *Salm.abortus-equi* H (e,n,x) serum. When tested with absorbed single-factor sera, the former colonies were agglutinated by pure v serum, the latter by pure x. By absorption it was found that a culture with the flagellar phase l,v completely removed all homologous agglutinins from *Salm.london* H (l,v) serum, and that *Salm. london* H (l,v) removed all homologous H (l,v) agglutinins from a serum made with the new organism. In addition, the flagellar phase H (e,n.x) of the new strain completely removed all these agglutinins from *Salm. abortus-equi* serum, and *Salm.abortus-equi* removed all the homologous agglutinins of phase H (e,n,x) from a serum made with the new organism. From a culture of Phase 1 in a medium containing the appropriate serum, Phase 2 was isolated ; similarly Phase 1 strains could be isolated from Phase 2 cultures. The flagellar antigens are therefore, l,v←→e,n,x.

Summary

A new *Salmonella* type has been isolated from the faeces of a baby suffering from acute gastro-enteritis. This organism has the antigenic structure XVI: l,v←→e,n,x and is designated *Salmonella salford*.

We wish to thank Dr. Kamiel who kindly supplied the clinical history of this patient.

A NEW TYPE OF *SALMONELLA* ISOLATED FROM PORCINE MESENTERIC LYMPH NODES—*SALM.NORWICH*

Joan Taylor, B.Sc., M.B., B.S., D.P.H, Salmonella Reference Laboratory, Colindale, A. Macdonald, M.D., and C. W. Sivell, F.I.M.L.T., Public Health Laboratory, Norwich.

During an investigation of porcine mesenteric lymph nodes an organism of the salmonella group was isolated from a pooled sample. This came from five healthy pigs which were slaughtered for pork. After incubation of the minced nodes in tetrathionate enrichment broth for 48 hours subcultures were made on deoxycholate citrate agar, and non-lactose-fermenting colonies typical of the salmonella group were isolated. This Gram-negative motile bacillus fermented dextrose, maltose, mannitol, dulcitol, sorbitol, xylose, trehalose and inositol with the production of acid and gas in 24 hours, and arabinose in 72 hours, but failed to ferment lactose, sucrose, salicin, adonitol or rhamnose. The citrate utilization test was positive, hydrogen sulphide was produced, but indole was not formed, urea was not hydrolysed, and there was no liquefaction of gelatin. The Voges-Proskauer test was negative and the methyl-red test positive.

On serological examination this organism appeared to have the antigenic formula VI.VII: 1,6. When tested with absorbed sera for factors 2, 3, 5, 6, 7, 10 it was agglutinated only by sera for factor 6. Many unsuccessful attempts were made to isolate colonies in H Phase 1 from the original culture which was in H Phase 2. When, however, the original culture was inoculated into soft agar containing antiserum made with *Salm.cholerae-suis* var. *kunzendorf,* an organism was isolated having the antigenic structure VI.VII: e,h.

A rabbit was immunized with the original culture which had been grown in broth and formolized. The antiserum agglutinated *Salm.oranienburg* 0 and the new strain 0 to 1:1,600; *Salm.birkenhead* H (1,6) and the new strain H (1,6) to 1 : 12,000; and *Salm.newport* H (e,h) and the new strain H (e,h) to 1 : 200. It was thought therefore that the new strain H (e,h) was the naturally occurring Phase 1 of this organism.

Reciprocal absorption tests using *Salm.oranienburg* 0 serum and a serum made with the new strain showed that absorption was complete; therefore the 0 complex was identified as VI.VII. Similarly, reciprocal absorption tests using *Salm.birkenhead* serum containing antibodies to H (1,6) and the serum made with the new strain showed that absorption was complete to H Phase 2 of both organisms. As the antiserum made with the original culture of the new organism gave a low titre to H Phase 1, a second serum was prepared using the new strain after passage through soft agar containing antibodies to *Salm.cholerae-suis* var. *kunzendorf*. This serum gave a titre of 1 : 6,400 to Phase 1 of the new strain and to *Salm.newport* H (e,h). Reciprocal absorption tests using the new serum and *Salm.newport* serum showed complete removal of agglutinins to Phase 1 of both organisms. Dr. P. R. Edwards states that he has received a similar strain which was isolated in the United States of America from a human source.

Summary

A new *Salmonella* type, *Salm.norwich,* having the antigenic structure VI.VII: e,h$\longleftrightarrow$1,6 is described. It was isolated from porcine mesenteric lymph nodes.

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health (C.H. 657),
Whitehall,
London, S.W.1.
Tel.: VICTORIA 8540. Extn. 538.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38, Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(11469) Wt. 8066—4418 3200 3/51 D.L. HE/4

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APRIL **1951** **Vol. 10**

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

LEPTOSPIRA CANICOLA IN A GIRL AND HER PUPPY.

George Chesney, M.D., D.P.H., Consultant Physician, Alderney Infectious Diseases Hospital, Poole.

When poliomyelitis is epidemic patients whose illnesses are not due to the virus of poliomyelitis are admitted to hospital with a diagnosis of poliomyelitis because of suggestive meningeal symptoms. Alston and Broom (1949) in a letter to The Lancet of August 13th noted that the meningeal form of leptospirosis may closely resemble mild poliomyelitis without paralysis, that infections with *Leptospira icterohaemorrhagiae* and *Leptospira canicola* are being recognised with increasing frequency in this country and that many of the cases occur in the autumn.

Among a number of such patients admitted to Alderney Infectious Diseases Hospital in 1950 as having poliomyelitis was a girl infected with *Leptospirosis canicola* who is the subject of this article, and whose case presented several interesting features.

Canicola fever was first recorded in Europe in 1931 by Klarenbeck and Schüffner, but the number of cases recognised in this country has been small, though there is reason to believe that the infection may be much more common than the small number of cases reported suggests. Interest has been recently stimulated by the report of Campbell *et alia* on six human cases recognised in Bristol during 1949.

The disease is caused by the *Leptospira canicola* which is of the same genus as *L. icterohaemorrhagiae* (Weil's disease). *L. canicola* is a parasite of the dog. It causes uraemic signs and ulcerative stomatitis in the dog, though the infection sometimes seems symptomless and " healthy " carriers are common. It is voided in the dog's urine. Occasionally man is infected.

The case now recorded is of interest because (*a*) the disease initially suspected by the family doctor was typhoid fever ; (*b*) it was then regarded as being either tubercular meningitis or poliomyelitis ; (*c*) subsequently the original diagnosis of typhoid fever was again favoured because the onset of the disease was twelve days after camping in the country where the water supply was not above suspicion, and because an agglutination titre of 1/250 for typhoid was obtained ; and (*d*), finally, serological evidence of *L. canicola* was obtained and the infection was traced to the patient's puppy.

The History of the Girl's Illness

A well-nourished and well-developed girl, P.M. aged 13, had been at a girl guide camp in the country from the 28th July to the 4th August, 1950, and on the 16th August, i.e., twelve days later (the incubation period of typhoid) she complained of headache, retired to bed and continued ill with fever, sweating, vomiting and diarrhoea until the 22nd August. She had vertigo and stiffness of the neck, was lethargic and swayed when walking to the toilet. She had no cold or sore throat. She was treated with sulphonamides. The family doctor had, as already stated, at first suspected typhoid but, after severe headache on the night of the 23rd August, the patient was admitted to a general hospital on the 24th and was suspected of having either tubercular meningitis or poliomyelitis. She had lumbar punctures done on the 24th and 25th August with the following results :—

White cells :	370 per cmm.	Lymphocytes :	70 per cent.
Sugar :	normal	Chlorides :	700 mg. per cent.
Protein :	No increase.		

On the 26th she was transferred to Alderney Hospital as a case of poliomyelitis. On admission to Alderney Hospital on the 26th August she was lethargic, but intelligent and co-operative. Her tongue was coated, appetite poor, mucous membranes were pale ; there was no cyanosis and no jaundice. Examination of her heart, lungs, and abdomen revealed no abnormality. The spleen was not palpable. Her pupils reacted to light and accommodation ; there was no evidence of any cranial nerve involvement ; no nystagmus ; some neck rigidity with a positive Kernig's sign. All the reflexes were present and the plantar reflex was flexor. The right knee jerk was sluggish. She had stiffness of the muscles of the neck, complained of muscular pain in the lumbar region, and had severe generalised headache. Her condition, however, was not typical of poliomyelitis and further investigation was undertaken to exclude any other possibilities.

On the 24th August before admission to Alderney Hospital she had had a temperature of 100·6° which fell to and remained normal until the 31st August, when it rose in staircase fashion reaching 104° on the 5th September, thereafter falling and reaching normal on the 11th.

On the 1st September at the beginning of the second rise in temperature, she was complaining of pain in the muscles of both legs but there were no symptoms of paralysis, though it was then thought that she might be suffering from poliomyelitis. She slept much. Examination of the nervous system showed no abnormality apart from a sluggishness of the right knee jerk.

Typhoid fever was now again suspected and on the 4th September a Widal was done with the following results :—

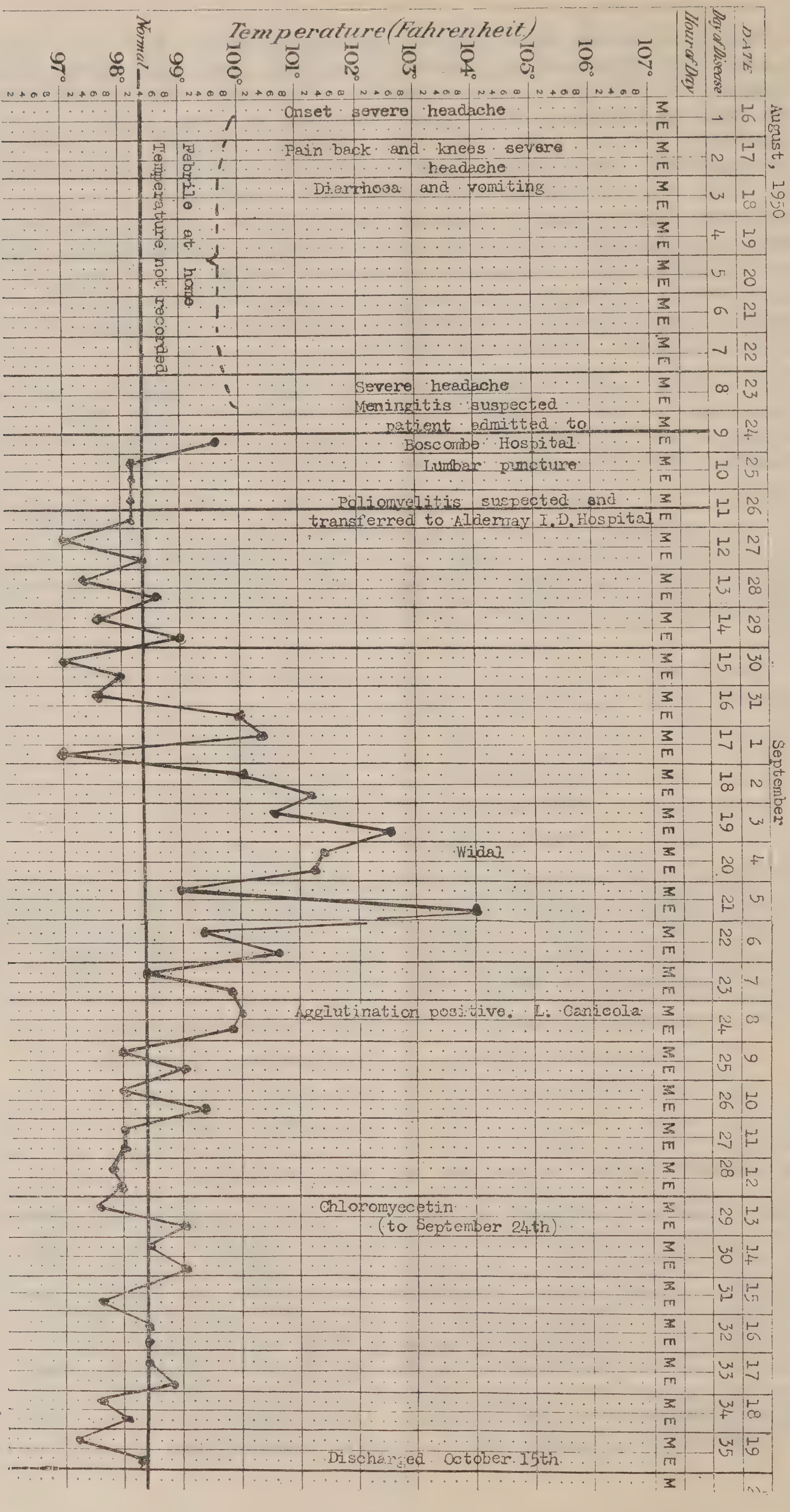
T.H. 1—250	T.O. negative
A.H. negative	A.O. negative
B.H. negative	B.O. negative
Abortus negative	

The bacteriologist reported that the agglutination tests were suggestive of typhoid fever, provided there had been no previous T.A.B. inoculations ; in fact she had never had T.A.B. vaccine. On the 4th September her urine showed a trace of albumin with pus cells, hyaline casts and epithelial cells. On 5th September her blood count was :—

R.B.C. 3,800,000	Polymorphs : 69 per cent.
Leucocytes : 6,800	Lymphocytes : 28 per cent.
Hb. 77 per cent.	Monocytes : 2 per cent.
	Eosinophils : 1 per cent.

The red cells showed anisocytosis and were macrocytic.

Her faeces were cultured on the 5th and 7th September but the results were negative for the typhoid, dysentery and salmonella groups ; the urine also was negative. The urine, however, still contained albumin, pus cells and scanty red blood cells. The Widal was repeated on the 7th September when again the titre for typhoid H. was 1/250, typhoid O. was negative, paratyphoid A.H. and O., paratyphoid B.H. and O. and abortus being negative. Faecal examinations on the 8th for the typhoid, dysentery and salmonella groups being again negative, a specimen of blood was sent on the 8th September to the Wellcome Laboratories of Tropical Medicine for examination for leptospirosis. A report received from Dr. J. C. Broom


Temperature (Fahrenheit)
97° 98° Normal 99° 100° 101° 102° 103° 104° 105° 106° 107°
DATE
Day of Disease
Hour of Day
August, 1950
September
Onset severe headache
Pain back and knees severe headache
Diarrhoea and vomiting
Febrile at home
Temperature not recorded
Severe headache
Meningitis suspected
patient admitted to Boscombe Hospital
Lumbar puncture
Poliomyelitis suspected and transferred to Alderney I.D. Hospital
Widal
Agglutination positive. L. Canicola
Chloromyecetin (to September 24th)
Discharged October 15th
M E

on the 13th September showed that the girl was undoubtedly suffering from canicola fever. The agglutination results were as follows:—

Leptospira icterohaemorrhagiae.	Agglutination	pos. at......1/300
	,,	neg. at......1/1,000
Leptospira canicola.	,,	pos. at......1/10,000
	,,	neg. at......1/30,000

On the 20th and 22nd September her urine was examined for *L. canicola* but none was found. Her urine was examined frequently and pus cells, hyaline casts and a trace of albumin persisted until the 11th October.

Treatment

Chloromycetin—one capsule of 0·25 gm. was given four-hourly from the 13th September to the 24th, a total of 48 gms., and penicillin 200,000 units twice a day was given from the 28th September to the 1st October (1,600,000 units).

The girl was discharged from hospital on the 15th October in good condition.

History of the Puppy's Illness

On the 1st May, 1950, a five weeks' old mongrel puppy was given to P.M. as a birthday present. He was quite healthy until early in July when he went off his food. He was seen by a veterinary surgeon who found that his gums were red and swollen. He vomited once or twice but recovered in a few days from his illness, which had been diagnosed as "teething" and he was apparently quite well before P.M. went to the girl guide camp on the 28th July. The girl was very fond of her puppy, and had nursed it when it was ill. It had frequently licked her face.

On the 19th September a specimen of blood was obtained from the dog, and Dr. Broom reported on the 22nd that agglutination was strongly positive for *L. canicola* at 1/1,000, negative at 1/3,000; agglutination for *L. icterohaemorrhagiae—positive:* 1/10 and negative at 1/30. There seems little doubt therefore that P.M. contracted her infection from the dog. Treatment with streptomycin was advised but considered impracticable, and the dog was destroyed.

Summary and Conclusions

A case of canicola fever is described. Two febrile phases occurred with a five-day remission. The illness suggested at certain stages tubercular meningitis, poliomyelitis and typhoid fever. The source of infection was a puppy that had had an illness diagnosed as "teething".

Owners of sick dogs should avoid the danger of infection from urine; licking of the face by healthy dogs is not without risk. In persons presenting obscure meningeal symptoms the possibility of meningeal leptospirosis should be borne in mind.

I wish to thank Dr. G. J. G. King of the Public Health Laboratory, Poole, for performing some of the laboratory investigations and Dr. J. C. Broom of the Wellcome Laboratories for the serological tests for leptospirosis.

SURVEY OF SICKNESS—SEPTEMBER QUARTER, 1950

(Issued from the General Register Office, Somerset House, W.C.2.)

The Registrar General's Quarterly Return No. 408 (in Tables A-H) gives detailed results of the Social Survey's interviews of random samples of the adult population carried out during the months August to November, 1950, in which the experience of July to September is recorded. The report given here presents some rates derived from these tables without standardization or correction for the number of days in the month, and compares them with those for preceding months. More detailed studies will appear in the publications of the General Register Office.

The Monthly Health Index—the proportion of persons, per 100 interviewed, who suffered no illness or injury so far as they remembered during the stated month—is given in Table A, separately for men and women, at ages 16-64 and ages 65 and over. The indices for men in both age groups and for elderly women were lower (worse) during July, August and September, 1950, than during the corresponding months of 1948 and 1949. The indices for women aged 16-64 were the same during July and August, 1950, as in the previous year.

Table B gives monthly morbidity rates (the proportion of persons, per 100 interviewed, who suffered at least one illness during the month, excluding those with injury only), days of incapacity and numbers of medical consultations, distinguishing persons with an illness commencing in the month from those with an illness continued from the previous month. Including persons with an injury average rates for the September quarters of 1948, 1949 and 1950 compare thus :—

	Ages 16–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
July–Sept., 1948 ...	61·7	73	35	80·5	150	57
July–Sept., 1949 ...	62·4	75	39	80·7	95	59
July–Sept., 1950 ...	64·1	68	38	83·7	100	67
July–Sept., 1950 per cent. of July–Sept.						
1948	104	93	109	104	67	118
1949	103	91	97	104	105	114

Compared with 1948 and 1949 rates of sickness were slightly increased at both ages during the September quarter of 1950. At ages 16-64 days of incapacity during the quarter were fewer than in July-September, 1948 and 1949 ; at ages 65 and over they were much fewer than in 1948 but slightly more than in 1949. Medical consultation rates at 16-64 declined slightly compared with a year before but were more than in July-September, 1948 ; among the elderly the frequency of medical consultation was greater than in either of the two preceding September quarters.

During the three months July, August and September, 1950, the proportion of persons aged 16-64 with new illness increased each month, whereas the proportion with continued illness declined. At both ages days of incapacity and medical consultation rates were higher in September than in July or August.

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during the month from those who developed an illness of a minor or ill-defined nature. Average monthly rates during the September quarters of 1948, 1949 and 1950 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	16–64		65 and over		16–64		65 and over	
	M	F	M	F	M	F	M	F
July–Sept., 1948	3·8	4·3	2·8	6·6	26·5	35·5	28·0	29·6
July–Sept., 1949	2·8	3·9	3·6	5·2	26·2	34·1	28·4	30·0
July–Sept., 1950	3·6	4·6	5·4	6·4	31·0	39·1	28·1	36·5

At ages 16-64 the morbidity rates from the more serious illnesses were higher in July-September, 1950, than in July-September, 1949, but much the same as in July-September, 1948 ; the rates for the minor conditions were higher than in either of the two previous September quarters. At ages 65 and over the rate of the more serious illnesses increased among males, whereas the rate of minor conditions increased among females.

During the course of the three months of the third quarter of 1950 almost all of the rates shown in Table C tended to fall in August but to increase again in September.

Table D gives average monthly numbers of new illnesses from selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the number of days of incapacity arising from each cause (including both new and continued illnesses).

The incidence of colds and influenza was higher during July-September, 1950, than in July-September, 1949, but a little lower than in July-September, 1948. The number of days of incapacity attributed to these complaints was higher than in either of the two previous September quarters.

The incidence of other respiratory disease and of rheumatism was generally higher than in July-September, 1950, than in the corresponding period of 1948 and 1949 but the days of incapacity were not increased.

Days of incapacity from all causes, shown in the bottom right-hand corner of Table D, increased among elderly men compared with the previous September quarter but among women and younger men the incapacity rates tended slightly to decline.

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 16–64						Ages 65 and over					
	M			F			M			F		
	1948	1949	1950	1948	1949	1950	1948	1949	1950	1948	1949	1950
January	40	36	34*	32	26	25*	21	20	21*	13	12	12*
February... ...	40	34	34	31	24	27	19	18	20	15	9	11
March	42	34	38	31	24	27	23	16	20	16	12	11
April	45	39	38	34	27	29	25	19	18	15	12	11
May	45	40	41	33	29	29	27	23	21	16	15	12
June	44	43	43	34	31	31	30	24	21	19	14	12
July	45	44	41	33	32	32	26	26	22	16	14	12
August	47	45	43	34	32	32	25	26	25	15	16	11
September ...	43	45	40	31	32	29	23	25	20	16	15	13
October	39	40		28	27		24	24		12	14	
November ...	36	36		27	27		22	23		12	13	
December ...	35	37*		25	28*		17	27*		12	14*	

(Based on combined results of interviews in two following months.)

* Experience based on one month's interviews corrected by factors based on previous experiences.

B.—*Monthly Morbidity Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 16–64				Ages 65 and over			
	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month
1948								
January ...	39·8	24·6	95	38	39·2	43·6	224	64
February ...	39·9	24·9	105	40	38·9	43·9	239	74
March ...	37·4	25·8	87	41	36·4	44·4	199	67
April	34·3	26·4	73	35	35·2	45·5	152	67
May	34·7	26·0	75	35	31·7	47·8	139	61
June	34·3	27·1	71	37	32·5	44·3	108	48
July	34·9	25·9	74	36	33·8	45·3	133	67
August ...	34·0	25·6	69	33	32·7	48·0	163	56
September ...	37·6	25·2	75	35	35·4	45·6	155	48
October ...	41·6	25·0	101	42	40·1	42·5	193	60
November ...	46·0	22·3	105	43	46·2	37·0	285	74
December ...	47·0	23·0	103	42	44·4	41·0	334	76
1949								
January ...	44·5	24·5	110	46	44·2	39·7	217	72
February ...	46·9	24·2	131	50	44·7	41·3	207	71
March ...	45·9	25·2	128	46	43·7	42·1	211	83
April	39·7	27·0	84	40	38·5	46·5	175	74
May	38·2	27·1	75	40	33·1	48·1	163	61
June	34·5	28·1	77	38	32·6	47·8	142	60
July	33·4	28·4	78	41	33·1	46·6	118	63
August ...	32·5	28·4	74	38	32·7	46·9	71	54
September ...	35·9	25·2	74	39	35·9	44·6	96	60
October ...	44·1	22·5	93	42	40·6	40·9	212	59
November ...	47·1	21·0	102	41	42·8	39·4	293	73
December* ...	51·0	20·1	100	41	46·3	36·5	323	65
1950								
January* ...	43·9	22·5	113	42	42·2	39·7	212	68
February ...	46·8	22·3	117	50	43·9	40·3	211	77
March ...	44·7	22·2	107	49	42·2	43·3	221	87
April	41·5	24·0	86	41	42·6	43·5	183	85
May	41·1	23·5	79	42	40·5	43·9	133	80
June	37·4	25·3	78	42	36·9	47·0	94	64
July	38·0	24·9	69	37	39·5	44·1	76	65
August ...	38·3	23·6	64	36	37·5	44·9	104	62
September ...	42·4	22·7	72	40	39·8	44·1	120	73
October ...								
November ...								
December ...								

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury, see Registrar General's Quarterly Returns, Table E.)

* Uncorrected rates based on one month's experience.

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
1948								
January ...	3·9	4·9	5·8	9·6	31·6	38·2	27·8	34·0
February ...	3·2	4·9	5·8	8·0	31·5	39·2	33·1	30·4
March ...	4·2	4·1	5·4	7·0	28·1	37·3	31·5	29·0
April	3·4	4·0	4·0	6·5	26·4	34·0	28·7	30·5
May	3·5	3·6	2·5	4·1	27·0	34·7	25·8	30·0
June	3·7	3·8	4·4	5·8	26·7	33·6	24·9	29·1
July	3·2	4·0	3·8	6·3	26·3	35·3	25·5	31·0
August ...	4·1	4·1	1·7	7·3	24·3	34·6	27·0	28·1
September ...	4·1	4·8	2·9	6·2	28·9	36·5	31·6	29·8
October ...	4·1	5·1	3·6	8·1	32·3	41·0	32·5	35·1
November ...	5·1	5·0	7·1	13·1	37·8	43·7	35·1	36·2
December ...	4·7	6·0	5·2	10·2	38·1	44·7	35·9	36·8
1949								
January ...	5·2	5·8	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	7·9	6·8	10·9	37·0	41·8	31·6	38·5
March ...	5·9	7·7	7·9	11·7	35·3	42·0	34·3	33·0
April	3·5	4·7	6·2	6·4	30·0	40·0	30·3	33·4
May	3·5	3·8	2·9	3·9	29·4	38·8	28·7	30·3
June	2·6	3·6	3·0	6·3	26·3	35·5	24·9	29·9
July	2·7	3·9	3·7	6·9	25·6	33·7	26·2	28·5
August ...	2·7	3·7	4·4	4·0	24·9	32·9	27·8	29·1
September ...	3·1	4·2	2·7	4·8	28·0	35·7	31·5	32·3
October ...	3·7	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·9	5·3	8·5	9·7	38·3	45·1	32·7	34·1
December* ...	4·5	6·6	5·4	9·7	42·3	47·9	32·4	42·2
1950								
January* ...	3·7	6·0	9·6	8·0	35·4	42·0	28·0	37·2
February ...	5·9	8·1	9·2	9·2	36·1	42·8	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·4	41·7	29·4	33·8
April	5·3	5·6	5·5	10·6	32·9	38·6	33·7	34·4
May	4·7	4·9	5·5	6·6	31·5	40·3	29·8	37·8
June	3·1	4·2	3·6	5·7	27·9	38·6	27·9	34·9
July	3·4	4·5	6·1	6·6	30·0	37·4	27·0	37·6
August ...	3·2	4·4	4·6	5·7	29·3	38·7	26·2	36·9
September ...	4·3	4·9	5·5	7·0	33·7	41·3	31·0	35·0
October ...								
November ...								
December ...								

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. " Ill-defined " excludes all symptomatic illness which caused incapacity for work, such cases being classed to the appropriate higher category. " Illnesses starting in each month " include new and recurrent illnesses.

* Uncorrected rates based on one month's experience.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity.*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1948								
January–March... ...	14·8	16·1	11·5	11·4	12·0	15·2	12·9	14·1
April–June	9·1	8·6	5·5	7·5	4·7	6·0	1·6	8·7
July–September... ...	9·8	9·3	6·1	6·7	3·3	5·3	1·9	3·4
October–December ...	20·0	20·3	12·2	13·9	12·9	16·6	9·3	23·9
1949								
January–March... ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September... ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March... ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September... ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...								
	Other respiratory disease							
1948								
January–March... ...	2·7	3·1	4·4	5·4	8·5	6·4	24·3	27·4
April–June	2·8	2·6	2·6	3·3	7·5	4·2	8·0	7·5
July–September... ...	1·8	2·7	2·7	2·6	3·3	2·4	10·1	9·6
October–December ...	3·6	3·3	4·8	4·9	9·5	7·3	24·2	22·0
1949								
January–March... ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September... ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March... ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September... ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...								
	Rheumatism, all forms							
1948								
January–March... ...	4·4	6·1	7·0	8·5	9·1	7·4	19·6	12·9
April–June	4·1	5·9	8·2	7·3	3·6	2·7	4·7	16·5
July–September... ...	4·3	6·6	8·1	7·1	4·9	4·4	9·2	5·9
October–December ...	5·5	7·8	9·9	10·7	5·1	4·6	3·7	20·9
1949								
January–March... ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September... ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March... ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	8·2	22·2
July–September... ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...								

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity*—(*continued*)

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Disorders of Heart and Arteries							
1948								
January–March... ...	0·3	0·9	1·5	1·9	3·3	2·7	0·9	18·1
April–June	0·3	0·6	1·7	1·6	2·0	2·8	9·8	11·1
July–September... ...	0·2	0·5	1·9	2·1	1·8	2·0	17·5	16·6
October–December ...	0·4	1·0	1·2	1·2	1·2	2·2	19·5	11·5
1949								
January–March... ...	0·4	0·7	0·6	1·1	5·2	3·7	10·7	6·4
April–June	0·1	0·4	0·5	1·0	3·4	2·6	10·4	5·2
July–September... ...	0·2	0·4	1·1	1·3	1·6	3·5	12·4	7·0
October–December ...	0·2	0·8	0·6	1·5	1·7	3·8	9·5	15·5
1950								
January–March... ...	0·2	0·6	1·3	1·3	3·7	4·2	12·5	14·6
April–June	0·3	0·7	0·5	1·5	4·8	2·9	13·9	19·1
July–September... ...	0·3	0·8	1·9	1·9	4·4	2·3	10·0	7·1
October–December ...								
	Skin complaints				All illnesses and injuries			
1948								
January–March... ...	1·5	1·1	2·0	0·6	110	85	203	235
April–June	1·8	1·2	0·7	0·9	85	63	128	136
July–September... ...	2·1	1·2	0·5	1·2	78	68	102	184
October–December ...	1·7	1·2	1·6	1·0	100	105	183	344
1949								
January–March... ...	1·3	1·4	0·4	0·9	119	127	200	220
April–June	1·7	1·4	0·8	0·9	84	74	151	165
July–September... ...	1·6	1·0	1·0	0·7	81	71	89	99
October–December ...	1·6	1·3	1·1	0·5	95*	98*	225*	302*
1950								
January–March... ...	1·6	1·5	1·4	1·0	111*	114*	196*	231*
April–June	1·9	1·4	0·8	0·8	88	75	143	133
July–September... ...	1·9	1·6	1·1	1·2	77	61	118	87
October–December ...								

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e., new, recurrent or continued from the previous month).

*Corrected for absence of February interviews.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

(Issued from the General Register Office, Somerset House, W.C.2.)

FEBRUARY, 1951

	Feb. 3rd	Feb. 10th	Feb. 17th	Feb. 24th	Average weekly figures for Feb., 1950
Scarlet Fever	875	892	904	828	2,005
Whooping Cough	4,916	5,114	5,662	5,930	1,644
Diphtheria	51	44	40	43	75
Measles, excluding Rubella	16,329	21,634	24,587	27,776	4,326
Acute Pneumonia	3,514	3,091	2,117	1,656	1,176
Meningococcal Infection	73	52	50	45	46
Acute Poliomyelitis (Paralytic) ...	21	12	18	16	27
,, ,, (Non-paralytic)	11	7	6	5	11
Ophthalmia Neonatorum	36	42	24	32	34
Puerperal Pyrexia and Puerperal Sepsis	87	90	67	81	89
Dysentery	967	985	972	1,366	333
Paratyphoid	3	6	11	13	5
Typhoid	3	5	4	5	7
Smallpox	—	—	1	—	—

No cases of Cholera or Plague or Typhus Fever.

MARCH, 1951

	Mar. 3rd	Mar. 10th	Mar. 17th	Mar. 24th	Mar. 31st	Average weekly figures for March, 1950
Scarlet Fever	952	1,056	1,009	927	918	1,875
Whooping Cough	6,099	5,637	5,527	4,081	4,098	2,003
Diphtheria	44	46	50	50	46	68
Measles, excluding Rubella ...	29,201	29,067	31,841	27,873	31,268	5,369
Acute Pneumonia	1,243	1,027	1,107	936	909	1,044
Meningococcal Infection	49	44	38	39	58	40
Acute Poliomyelitis (Paralytic) ...	19	9	18	10	19	23
,, ,, (Non paralytic)	9	4	—	3	4	9
Ophthalmia Neonatorum ...	57	48	41	34	20	36
Puerperal Pyrexia and Puerperal Sepsis	69	86	80	65	62	101
Dysentery	1,364	1,449	1,672	1,257	999	367
Paratyphoid	2	4	5	10	21	5
Typhoid	4	11	1	7	7	7
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

THE ROUTINE USE OF SELECTIVE AND ENRICHMENT MEDIA FOR THE ISOLATION OF SALMONELLAE

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During the period September, 1946, to January, 1950, organisms of the Salmonella group were cultivated from approximately 600 specimens of faeces, 539 of which were examined by a standard method. It was thought that this number of positive specimens was sufficiently large for an attempt to be made to assess the relative efficiency of plating directly and after enrichment, and to compare the two plating media and the two liquid enrichment media used.

Fifteen types of salmonellae were isolated during this period. *Salm. typhi-murium* was isolated on 309 occasions and the next most frequently isolated organism was *Salm. paratyphi B* (73 occasions); *Salm. typhi* was isolated from 27 specimens.

Media

The media used were prepared by standard methods throughout the period of the investigation, though a considerable number of batches of each medium were necessarily employed.

They were as follows:—

(1) Hynes' (1942) modification of Leifson's deoxycholate-citrate agar;

(2) Tabet's (1938) modification of Wilson and Blair's medium;

(3) Hobbs and Allison's (1945*a*) modification of selenite-F broth;

(4) a modification of Kauffmann's (1935-36) tetrathionate broth. This medium contained only 60 per cent. of the brilliant green originally used by Kauffmann.

Cultural methods

A large loopful of the faeces, if liquid, or of a broth suspension of the faeces, if solid, was inoculated direct on to deoxycholate-citrate agar (Method DDC), on to Wilson and Blair's medium (Method DWB), into a modification of Kauffmann's tetrathionate broth and into selenite-F broth. After overnight incubation the tetrathionate broth was plated on to deoxycholate-citrate agar (Method TDC) and Wilson and Blair's medium (Method TWB) and the selenite broth was plated on to the same two solid media (Methods SDC and SWB). The deoxycholate-citrate agar plates were examined after 24 hours' incubation and the Wilson and Blair plates after 24 and 48 hours. Non-lactose-fermenting colonies on deoxycholate-citrate agar and suspicious colonies on Wilson and Blair's medium were investigated biochemically, either in peptone water sugars or by means of a screening plate (Knox 1949) and when necessary serologically.

All salmonella strains isolated for the first time from any patient were identified by full biochemical and serological tests. With subsequent isolations serological and biochemical confirmation were generally employed with strains from all media, though in a number of cases where salmonella organisms had been identified on deoxycholate-citrate agar, typical colonies on Wilson and Blair's medium were accepted as sufficient evidence of the specimen being positive on this medium also.

Results

Five hundred and thirty-nine specimens were positive by one or more methods, of which 176 were positive by all six methods. Table I shows the number of specimens positive by each direct and indirect method. In Tables I, II, III and IV and in the comparisons which follow in the first part of the paper the results with all types of salmonellae, including *Salm. typhi,* have been considered. Although *Salm. typhi* was isolated from only 27 specimens, the results with this organism differed in some respects from the others and are considered separately later in the paper.

TABLE I

Number of positive salmonella results (including Salm. typhi) *obtained from* 539 *specimens by each of the two direct and four indirect plating methods.*

ENRICHMENT MEDIUM	PLATING MEDIUM		
	Number positive on deoxycholate-citrate agar	Number positive on Wilson and Blair's medium	Total number positive on both media
NONE (Direct plating methods DDC and DWB)	320 (59·4)	313 (58·1)	633 (58·7)
TETRATHIONATE BROTH (Indirect plating methods TDC and TWB)	437 (81·1)	406 (75·3)	843 (78·2)
SELENITE BROTH (Indirect plating methods SDC and SWB) ...	462 (85·7)	421 (78·1)	883 (81·9)
TETRATHIONATE BROTH AND SELENITE BROTH (Indirect plating methods TDC, SDC, TWB and SWB) ...	899 (83·4)	827 (76·7)	1,726 (80·1)

Percentages in parentheses.

Comparison of deoxycholate-citrate agar and Wilson and Blair's medium

(a) *Direct plating*

A comparison of the two direct methods is shown in Table II.

Of the 539 specimens positive by one or more methods, 396 (73·5 per cent.) were positive on direct plating. Two hundred and thirty-seven (44·0 per cent.) were positive by both the direct methods, DDC and DWB, 83 (15·4 per cent.) were positive by Method DDC only, and 76 (14·1 per cent.) were positive by Method DWB only. One hundred and forty-three (26·5 per cent.) specimens were negative by both direct methods.

An almost equal number of positive results (nearly 60 per cent. of the total number positive) was obtained by each direct method, and the omission of either would have resulted in missing approximately one in five of all specimens positive on direct plating.

TABLE II

Comparison of the two direct plating methods in the isolation of 539 *salmonellae (including* Salm. typhi).

Direct plating method DWB	Direct plating method DDC		TOTAL
	Positive	Negative	
Positive	237 (44·0)	76 (14·1)	313 (58·1)
Negative	83 (15·4)	143 (26·5)	226 (41·9)
TOTAL	320 (59·4)	219 (40·6)	539 (100)

Percentages in parentheses.

(b) *Indirect plating*

A comparison of deoxycholate-citrate agar and Wilson and Blair's medium as indirect plating media is shown in Table I. Deoxycholate-citrate agar gave 31 more positives than Wilson and Blair's medium when plating from tetrathionate broth and 41 more positives when plating from selenite broth. In each instance the increase obtained on deoxycholate-citrate agar is significant.

One hundred and forty-three specimens were positive only on indirect plating and of these 29 and 15 would have been missed if deoxycholate-citrate agar or Wilson and Blair's medium respectively had been omitted as an indirect plating medium. (Table III.)

TABLE III

Comparison of enrichment media and plating media among 65 specimens positive by one or two indirect methods only.

ENRICHMENT MEDIUM	PLATING MEDIUM			
	Number positive on deoxycholate citrate agar only	Number positive on Wilson and Blair's medium only	Number positive on both deoxycholate citrate agar and Wilson and Blair's medium	
TETRATHIONATE BROTH (methods TDC and TWB) ...	7	5	9	21*c*
SELENITE F BROTH (methods SDC and SWB)	14	5	12	31*d*
BOTH TETRATHIONATE AND SELENITE F BROTH (methods TDC, SDC, TWB and SWB) ...	8	5		
	29*a*	15*b*		

(*a*) Total number positive on deoxycholate citrate agar only (methods TDC and SDC).
(*b*) Total number positive on Wilson and Blair's medium only (methods TWB and SWB).
(*c*) Total number positive after enrichment in tetrathionate broth only (methods TDC and TWB).
(*d*) Total number positive after enrichment in selenite broth only (methods SDC and SWB).

One hundred and forty-three specimens were positive on indirect plating only. Sixty-five are considered above; of the other 78, one was positive by methods TDC and SWB, and the remainder were positive by three or four methods.

Comparison of tetrathionate broth and selenite broth

Table I shows the number of positive results obtained from each of the two liquid enrichment media when plated on to the same solid medium. Method SDC gave 85·7 per cent. positives as compared with 81·1 per cent. by Method TDC, and Method SWB 78·1 per cent. as compared with 75·3 per cent. by Method TWB. The increase obtained by plating from selenite broth is significant in each instance.

Of the 143 specimens positive only on indirect plating 21 and 31 would have been missed if tetrathionate broth or selenite broth respectively had been omitted. (Table III.)

Comparison of direct and indirect plating

Six hundred and thirty-three (58·7 per cent.) positive results were obtained from 1,078 direct platings compared with 1,726 (80·1 per cent.) positive results from 2,156 indirect platings. (Table I.)

In Table IV the results obtained by Methods DDC and DWB have each been divided into three categories.

TABLE IV

Number of positive salmonella results (including Salm. typhi) *obtained from* 539 *specimens by the four indirect plating methods for each of the six categories of direct plating.*

DIRECT			INDIRECT				
			Number positive by				
Method	Category	Number	Method TDC	Method SDC	Method TWB	Method SWB	One or more indirect methods
DDC	Heavy positive	183	178(97·3)	173(94·5)	162(88·5)	163(89·1)	183(100·0)
DWB	+ +	123	114(92·7)	112(91·1)	114(92·7)	116(94·3)	123(100·0)
DDC	Light positive ...	137	110(80·3)	118(86·1)	96(70·1)	105(76·6)	132(96·4)
DWB	− +	190	163(85·8)	168(88·4)	157(82·6)	155(81·6)	185(97·4)
DDC	Negative ...	219	149(68·0)	171(78·1)	148(67·6)	153(69·9)	214(97·7)
DWB	− −	226	160(70·8)	182(80·5)	135(59·7)	150(66·4)	221(97·8)
TOTAL	NUMBER POSITIVE	539	437(81·1)	462(85·7)	406(75·3)	421(78·1)	529(98·1)

Heavy positive = 50 per cent. or more of total growth on plate.
Light positive = less than 50 per cent. of total growth on plate.
+ + = positive after 24 and 48 hours' incubation.
− + = negative at 24, but positive after 48 hours' incubation.

Percentages in parentheses.

Method DDC

1. Heavy positive (50 per cent. or more of total growth on plate).
2. Light positive (less than 50 per cent. of total growth on plate).
3. Negative.

Method DWB

1. Positive after 24 and 48 hours' incubation (+ +).
2. Negative at 24 hours' and positive only after 48 hours' incubation (− +).
3. Negative after both periods of incubation (− −).

The table shows the number of positive results obtained by the four indirect plating methods for each of these six categories of direct plating. As would be expected the largest percentage of positive results on indirect plating was obtained with the heaviest grades of growth on deoxycholate-citrate agar and Wilson and Blair's medium. It is, however, of interest that, whereas with specimens positive on direct plating the percentage of positives obtained by each of the indirect methods was approximately equal, with specimens negative on direct plating (and therefore presumably with specimens containing relatively few organisms) there is a significant superiority of selenite broth (Methods SDC and SWB) over tetrathionate broth (Methods TDC and TWB).

Results with Salm. typhi

Salm. typhi was isolated from 27 specimens, four of which were positive by all six methods.

The main differences between the results with *Salm. typhi* and those with the other groups of salmonellae are as follows :—

1. The superiority of Wilson and Blair's medium over deoxycholate-citrate agar as a direct plating medium (Table V). Nineteen of 27 specimens (70·4 per cent.) gave a positive result by Method DWB as compared with only 11 (40·7 per cent.) by Method DDC, a difference which is just significant. The corresponding percentages for the whole group were 58·1 and 59·4 (Table I). With 4 of the 27 specimens, *Salm. typhi* was isolated from the direct Wilson and Blair plate only.

2. The decreased usefulness of indirect plating in the isolation of *Salm. typhi.* Thirty positive results were obtained from 54 direct platings (55·6 per cent.) compared with 67 positive results from 108 indirect platings (62 per cent.) (Table V). The corresponding percentages for the whole group were 58·7 and 80·1 (Table I).

TABLE V

Number of positive Salm. typhi *results obtained from 27 specimens by each of the two direct and four indirect plating methods.*

ENRICHMENT MEDIUM	PLATING MEDIUM		
	Number positive on deoxycholate-citrate agar	Number positive on Wilson and Blair's medium	Total number positive on both media
NONE (Direct plating methods DDC and DWB)	11 (40·7)	19 (70·4)	30 (55·6)
TETRATHIONATE BROTH (Indirect plating methods TDC and TWB)	12 (44·4)	16 (59·3)	28 (51·9)
SELENITE BROTH (Indirect plating methods SDC and SWB) ...	19 (70·5)	20 (74·1)	39 (72·2)
TETRATHIONATE BROTH AND SELENITE BROTH (Indirect plating methods TDC, SDC, TWB and SWB) ...	31 (57·4)	36 (66·7)	67 (62·0)

Percentages in parentheses.

Discussion

Results obtained from a comparison of different selective and enrichment media may be influenced by a number of factors ; the difficulty of assessing their relative merits has been discussed by Knox and his colleagues (1942). Even if such comparisons are made on identical specimens, as in this survey, the number examined should be large enough to be representative of all types of cases and carriers and include a variety of salmonella types. Batch variation of the media is unavoidable in most routine laboratories, though this is more liable to happen with some media than with others. The media employed throughout this investigation were those used for routine work and it was thought that a sustained variation between batches of any particular medium would be reflected in the results. It should be emphasized that our results are based on media made up by methods to which reference has been made. A number of modifications of these media are in use in other laboratories and it is not suggested that these would necessarily give results similar to our own.

In general, deoxycholate-citrate agar proved superior to Wilson and Blair's medium both directly and after enrichment. This was not so, however, for *Salm. typhi* ; our results with this organism are similar to those of Hobbs and Allison (1945*b*), who obtained more positive results with Wilson and Blair's medium than with deoxycholate-citrate agar, both directly and after enrichment.

Salmonella organisms of all types were isolated from approximately 33 per cent. more specimens after enrichment than on direct plating, and 25-40 per cent. more positive results were obtained by the use of any of the indirect plating methods.

With *Salm. typhi,* however, the superiority of enrichment methods over direct plating was dependent on the combination of media employed. For instance, with Wilson and Blair's medium the percentage of positive results on direct plating was very little less than that after selenite enrichment, though with deoxycholate-citrate agar approximately 75 per cent. more positive results were obtained after growth in selenite broth. No significant advantage was obtained from tetrathionate enrichment with either of the two solid media. Similar results were obtained by Knox, Gell and Pollock (1942) using MacConkey agar, eosin-brilliant green-methylene blue-agar and tetrathionate agar as direct plates and a tetrathionate broth approximating to Muller's as the liquid enrichment medium.

Of the two liquid media, selenite broth proved superior to tetrathionate broth both for the whole group and for *Salm. typhi* irrespective of the indirect plating medium used. In this connection it may be noted that the tetrathionate broth used throughout this investigation contained an excess of thiosulphate ; although rather more selective, a tetrathionate medium of this type is more inhibitory to *Salm. typhi* than a " balanced " medium (Knox *et al.* 1943). In spite of the presence of brilliant green, growth of proteus strains may still occur in our tetrathionate medium, and this may partly account for the inferiority of tetrathionate broth to selenite broth as a selective enrichment medium during our investigation.

With *Salm. paratyphi B,* however, in contrast with the other salmonella strains, the number of positive results obtained by Method SWB was significantly less than that by Methods SDC, TDC or TWB. The satisfactory results by Method SDC suggest that *Salm. paratyphi B* grows equally well in selenite broth and tetrathionate broth, and the explanation of the poor results on SWB may lie in an increased susceptibility of *Salm. paratyphi B* to the combined inhibitory effect of selenite and Wilson and Blair's medium. Inhibition of growth in the inoculated area is frequently seen when selenite broth cultures of salmonella organisms are plated on to Wilson and Blair's medium, though satisfactory colonies are obtained towards the edge of this area where the concentration of selenite on the plate is presumably much less. There were sixteen specimens from which *Salm. paratyphi B* was isolated on deoxycholate-citrate agar after selenite enrichment and not on the corresponding Wilson and Blair plate and it is of interest that fifteen of the sixteen negative Wilson and Blair plates were sterile.

The most successful method was plating from selenite broth to deoxycholate-citrate agar (Method SDC) ; its superiority was most pronounced with specimens negative on one or other of the direct plating media. The value of this procedure has also been shown by Cruickshank and Williams Smith (1949) during an investigation of dogs, cats and pigeons for salmonellae. It is evident, however, from our results that the larger the number of selective and enrichment media used, the greater is the chance of isolating salmonella organisms from specimens of faeces. If deoxycholate-citrate agar, Wilson and Blair's medium, tetrathionate broth or selenite broth had been omitted the respective percentages of positive specimens missed would have been 6·1, 3·7, 3·9 and 5·8.

In routine work we have found Wilson and Blair's medium a valuable confirmatory medium in addition to deoxycholate-citrate agar. Although its behaviour may be variable, attention is quickly drawn to the characteristic appearances of salmonella colonies and the medium has been found of particular value in mixed salmonella and proteus infections. As our results and those of other workers have shown, Wilson and Blair's medium is specially useful in the isolation of *Salm. typhi* and whenever possible it should be included in the cultural routine, particularly during outbreaks of typhoid fever.

Summary

A comparison of deoxycholate-citrate agar, solid Wilson and Blair's medium, tetrathionate broth and selenite-F broth for the isolation of salmonella organisms has been made on 539 positive specimens of faeces.

For the group as a whole (including *Salm. typhi*) deoxycholate-citrate agar gave more positive results than Wilson and Blair's medium. Of 539 specimens examined, 320 (59·4 per cent.) were positive on deoxycholate-citrate agar on direct plating compared with 313 (58·1 per cent.) on Wilson and Blair's medium (Table II). On indirect plating 899 (83·4 per cent.) out of 1,078 platings on deoxycholate-citrate agar were positive compared with 827 (76·7 per cent.) on Wilson and Blair's medium (Table I). Selenite broth proved superior to tetrathionate broth as an enrichment medium. Out of 1,078 platings from each enrichment medium 883 (81·9 per cent.) were positive from selenite broth compared with 843 (78·2 per cent.) from tetrathionate broth (Table I). In general however, the results showed that the use of as many selective and enrichment media as possible provides the best chance of a positive finding.

The results with *Salm. typhi* differed in some respects from those obtained with the whole group and confirmed the special value of Wilson and Blair's medium for the isolation of this organism. On direct plating 19 (70·4 per cent.) of 27 specimens were positive on Wilson and Blair's medium compared with only 11 (40·7 per cent.) on deoxycholate-citrate agar. Indirect plating has less advantage with *Salm. typhi* than with the group as a whole. Thirty (55·6 per cent.) positive results were obtained from 54 direct platings compared with 67 (62 per cent.) from 108 indirect platings (Table V). The corresponding percentages for the group as a whole were 58·7 and 80·1 (Table I).

We should like to thank Professor Robert Knox for his interest and many helpful suggestions during this investigation, and Dr. Ian Sutherland (Institute of Social Medicine, Oxford) for his statistical advice. We should also like to express our indebtedness to the Nuffield Bureau of Health and Sickness Records, Oxford, for their help in extracting the information necessary for compiling the tables.

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SALMONELLA FOOD POISONING DUE TO DUCK EGGS

with

a trial of Chloramphenicol on the Carrier State

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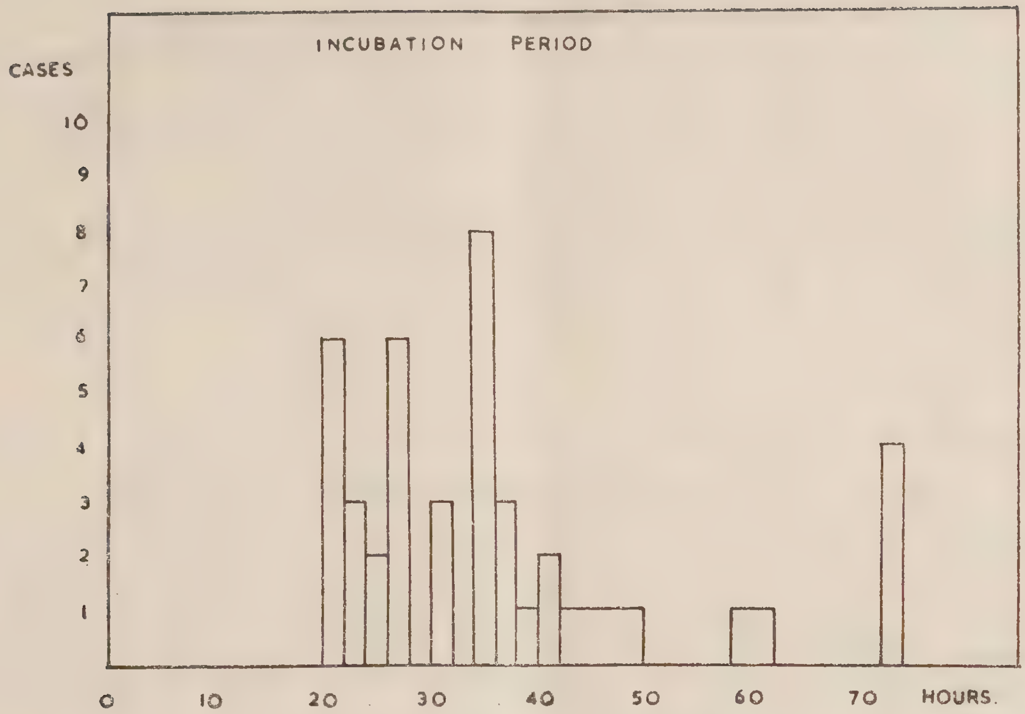
An outbreak of food poisoning occurred suddenly in April, 1950, among the nursing and auxiliary staff serving a group of teaching hospitals in Belfast. Because of its severity, extent and probable cause, this outbreak is considered worthy of record. It affected some sixty-eight persons, either nurses or kitchen maids, and resulted in much administrative difficulty. The pay block hospital in the group had to be temporarily closed because of the large number of nurses from this hospital that developed food poisoning. The vehicle of infection was proved to be a lemon meringue pie, which formed part of an evening meal, and the presumptive source of infection was the duck eggs used in the manufacture of the pie. An attempt to trace the source of the duck eggs met with no success, as it led to a central collecting station, where no record was kept of the farms from which the eggs originated.

The Outbreak

On Wednesday, 19th April, 17 nurses reported sick, and by Friday, 21st April, the number had risen to 44. The predominating symptoms were nausea, vomiting and diarrhoea associated, in some cases, with abdominal pain. Pyrexia, ranging up to 103° F. and persisting for a maximum of four days, was present in 31 cases (70 per cent.). Laboratory investigations revealed that the infecting organism was *Salm. typhi-murium ;* this organism was isolated from 40 out of 44 cases. A careful dietary history of all the affected staff was taken. This showed that a lemon meringue which had been served as part of the evening meal on the day preceding the outbreak was an item common to the diet of all cases. In some instances nurses returning from " off-duty " had not partaken of the meal, but had portions of the lemon meringue pie only. The pie was prepared during the morning of 18th April in the kitchen of the Nurses' Home, the yolks and whites of eighteen duck eggs being used. The yolks were added to a mixture of cornflour, lemon juice, and sugar, prepared by mixing these ingredients to a paste with water which had been brought to the boil and allowed to cool slightly. Previously prepared pastry was covered with this mixture, which was allowed to set at room temperature. The meringue was prepared by whipping the whites of the duck eggs and spreading them on top of the tart, which was then placed in a cool oven for half an hour to dry. At no stage were the eggs subjected to any cooking other than this. As is so common when investigating a food-borne outbreak, none of the suspected pie remained for bacteriological examination. The possibility of some member of the kitchen staff being a carrier and source of the outbreak was explored. It was found that no member of the kitchen staff, apart from those who had partaken of the lemon meringue pie, had any salmonellae in their stools. This confirmed our opinion as to the source of infection.

The incubation periods for the 44 cases of gastro-enteritis have been plotted (Fig. 1). It will be seen that the incubation period varied from 20 to 72 hours, but that in most cases the time of onset fell within the period 20 to 50 hours.

Figure 1



Laboratory Investigations

Methods

All specimens of stool were subjected to preliminary enrichment in selenite F medium for 18 hours and then plated out on to Hynes' modification of Leifson's sodium deoxycholate-citrate medium. Non-lactose-fermenting colonies were primarily identified by fermentation reactions and slide agglutination tests with salmonella antisera (Standards Laboratory) diluted one in five. Later the fermentation tests were omitted and slide agglutination of pale colonies picked off the deoxycholate plates was alone used in identification. The primary enrichment resulted in a very high isolation rate. Direct plating of faeces on to deoxycholate medium was frequently negative when enrichment resulted in a good growth, so that direct plating was discontinued to save time and materials. Figures on the advantage of enrichment over direct plating are therefore not available. The routine procedure was to examine three consecutive daily specimens of stool, those persons giving a positive culture being followed for a period of time varying from 14 to 26 days.

Results

When it was discovered that the outbreak was one of salmonella food poisoning, it was decided to investigate all the nursing and auxiliary staff who had eaten the incriminated evening meal. As already mentioned, *Salm. typhi-murium* was isolated from 40 out of 44 clinical cases. Three consecutive daily specimens of stool were now examined from a further 45 persons and as a result 28 asymptomatic carriers were discovered. Strict isolation was enforced on all cases giving a positive stool culture. No salmonellae were isolated from the remaining 145 nurses who occupied the same nurses' home but who had not eaten any of the meringue pie. Thus, observations over a period of 17 days after the outbreak failed to reveal the presence of any secondary cases. This may well have been the result of the strict isolation enforced, as secondary cases are a common finding in outbreaks of salmonella food poisoning. From Table 1 it is seen that 44 persons out of 89 at risk were affected, an attack rate of 49·4 per cent., and that there were 28 asymptomatic carriers (31·5 per cent. of the persons at risk).

TABLE 1

The Isolation of Salmonellae from Staff at Risk

	Number	*Salm. typhi-murium* in stools
With symptoms	44	40
Without symptoms	45	28
Total	89	68

Trial of Chloramphenicol for clearance of excreters

The onset of the gastro-enteritis was abrupt, and the clinical course of the disease was short. The majority of patients recovered within 48 hours. The return to duty of those nurses with positive cultures of *Salm. typhi-murium* in their stools presented a problem, and as a policy of safety it was decided to exclude from the wards all the affected staff until three consecutive daily specimens of stool were negative.

In an attempt to speed the disappearance of salmonellae from the stools it was decided to try the effect of giving three grams of chloramphenicol in divided doses for three days to ambulant cases and carriers. Accordingly 50 nursing and auxiliary staff excreting *Salm. typhi-murium* in their stools, irrespective of whether they were clinical cases or asymptomatic carriers, were divided into two groups by taking alternate names from a list. One group was given chloramphenicol and the other acted as a control. Specimens of stool were examined daily (or as near daily as possible) for a period of three weeks or more. Table 2 shows the rate of disappearance of *Salm. typhi-murium* from the stools of the two groups. It can be seen that in the majority of cases the stools were negative within fourteen days (82 per cent.), but a few were more persistent convalescent carriers. In two cases positive cultures were still obtained on the twenty-first day, but both were negative by the thirtieth day.

TABLE 2

Duration of Excretion of Salm. typhi-murium *in untreated Persons and in Persons treated with Chloramphenicol*

Weeks	Excreters yielding negative stool cultures		
	Treated	Untreated	Total
0 – 1	8	7	15
1 – 2	12	14	26
2 – 3	3	4	7
> 3	2	0	2
Totals	25	25	50

The number of observations is not very great. The figures were therefore rearranged in the form of a four-fold table, which showed that there was no significant statistical difference between the two groups. This negative result merely proves that chloramphenicol in the dosage administered had no effect on the rate of clearance of *Salm. typhi-murium* from the stools. It is realized that this dosage was small in comparison with that advocated by various authors for the treatment of acute cases of salmonella infection, but this experiment was only a tentative one with the purely utilitarian object of trying to render the nursing staff free from infection in the shortest possible time. It can only be regarded as a failure.

Experimental Reproduction of Cooking Procedure

In order to observe the survival of *Salm. typhi-murium* in dishes of this description, artificially infected materials were used to prepare a lemon meringue pie in the laboratory by the method employed by the cook in the nurses' home. It was also proposed to use other times and temperatures which appear to be more normal for the preparation of this dish. A preliminary experiment showed that the salmonellae tended to survive in the yolk mixture rather than in the overlying meringue made with the egg white, and further experiments were limited to the testing of egg yolk and cornflour mixtures artificially infected with small or large inocula of an 18-hour broth culture of *Salm. typhi-murium* and heated in an oven for various times at different temperatures.

The results of these experiments are recorded in Table 3, which for simplicity shows degrees of contamination in place of the actual numbers of organisms per gram of food, as determined by the plate count.

TABLE 3

Survival of Salm. typhi-murium *in artificially contaminated Egg-Yolk and Cornflour mixtures*

			After subsequent heating in the oven		
	Cold Mixture	Mixture after addition of boiling water and cooling	A 100° C. for 10 minutes	B 100° C. for 10 minutes followed by 60° C. for 20 minutes	C 100° C. for 30 minutes
Small inoculum	++++	++	+	—	—
Large inoculum	++++	++	++	++	—

+ to ++++ = degrees of contamination.

Method B was used for the preparation of the pie causing this outbreak, whereas the method normally used in the hospital kitchen as a quick technique is C. In normal domestic practice the pie is cooked at about 80-90° C. for three hours. It will be seen that *Salm. typhi-murium* when present in large numbers in an egg mixture will survive the addition of boiling water and further cooking in the oven at 100° C. for ten minutes followed by 60° C. for twenty minutes.

Discussion

This outbreak of food poisoning due to *Salm. typhi-murium* occurring among nursing staff resembles that described by Garrod and McElroy (1949), although it was on a much smaller scale. In the outbreak described by these workers about 136 members of the nursing and domestic staffs of a large hospital were affected and, as the same meal was served in the wards, many patients were also affected. In the outbreak now described, only the nursing and domestic staffs were affected, as the wards were served with their meals from an entirely separate kitchen. The attack rate was only 49·4 per cent., which is significantly lower than that noted by Garrod and McElroy (69 per cent.). However, the infection rate after ingestion of the infected food was 76·4 per cent.

The vehicle of infection was considered to be a lemon meringue pie made with duck eggs ; these are known to be a potential source of danger. Many cases of salmonella food poisoning due to duck eggs have been previously described (Gordon and Buxton 1945, Gillespie 1946, Garrod and McElroy 1949) in which the infecting organism was *Salm. typhi-murium.*

The effect of cooking on the destruction of salmonellae in eggs has received considerable attention since Rettger, Hull and Sturges (1916) first infected eggs with *Salm. pullorum* and found that boiling for four minutes did not always destroy the organisms but that, if the eggs were poached or scrambled, the infecting organism was rapidly killed. Subsequent work on reconstituted dried egg by Gibbons and Moore (1944) and Haines, Elliot and Tomlinson (Report 1947) showed that scrambled egg, custards, and cake were usually rendered sterile by normal cooking procedures. However, if the dose of salmonellae present before cooking was large, either from incubation of the food overnight or from artificial inoculation, then cooking could not be relied on to destroy them all (Report 1947).

The most recent work of Clarenburg and Burger (1950) indicates that large duck eggs (80 gm.) cannot be rendered safe by boiling in less than 14 minutes, if they are to be eaten at once, though 10 minutes' boiling is sufficient if they are left for a further 5 minutes after being removed from the pan. To ensure the destruction of salmonellae a temperature of 59-60° C. must be attained in the centre of the egg. It would therefore appear that no method of cooking can be recommended that does not result in coagulation of the egg yolk. Ordinary poaching is clearly inadequate.

The lemon meringue pie which was the vehicle of the present outbreak was prepared with the minimum amount of heat for palatability, i.e. 100° C. for ten minutes followed by 60° C. for twenty minutes. As there is so much variation in the degree of heat which various cooks employ for egg dishes it would appear to be unwise to continue to use duck eggs for this purpose until such time as they can be guaranteed to be reasonably free from infection at the source.

Summary

An outbreak of food poisoning due to *Salm. typhi-murium* among the nursing and auxiliary staff of a hospital is described. The vehicle of infection was a lemon meringue pie which was eaten by 89 persons, of whom 44 (49·4 per cent.) were affected. Twenty-eight asymptomatic excreters were discovered.

Duck eggs used in the preparation of the pie were the probable source of infection. Experiments showed that salmonellae, if present in large numbers in an egg yolk and cornflour mixture, could survive the heating conditions of preparation, namely 100° C. for ten minutes followed by 60° C. for twenty minutes. It is emphasized that no method of cooking duck eggs can be recommended that does not result in coagulation of the yolk.

In a small controlled trial, chloramphenicol in a dosage of one gram daily for three days did not hasten the disappearance of *Salm. typhi-murium* from the stools of infected persons.

Our thanks are due to Dr. M. S. Campbell, Medical Officer to the Nursing Staff, Royal Victoria Hospital, for much assistance during this investigation.

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THE INCIDENCE OF DENTAL CARIES AMONG ADULTS AND YOUNG CHILDREN IN THREE HIGH- AND THREE LOW- FLUORINE AREAS IN ENGLAND

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Reports have come from many parts of the world of a low incidence of dental caries in communities living in areas where the drinking water has a high fluorine content. Caries experience has been found to decrease as the amount of fluorine increases (Dean, Arnold Elvove, 1942) but with concentrations above about 1·5 parts per million mottling of the teeth, the first symptom of fluorosis, becomes increasingly apparent. With a concentration of 5 parts per million teeth were found to be seriously disfigured (Ainsworth, 1933) and with still higher amounts toxic symptoms and bone lesions may occur. A concentration of 1·0 to 1·2 parts per million of fluorine gives a considerable reduction in caries without appreciable mottling: any which might occur is so mild as to escape notice unless the teeth are specifically examined for the condition.

It has been found that if the ingestion of fluorine is to have an effect on the teeth it must take place during the period of tooth formation, that is, during the first eight years of life. After this age and after eruption it has little effect (Klein, 1948).

The caries experience of twelve year old school children born and brought up in a high fluorine area in this country was found to be only half that of children in a low fluorine area (Weaver, 1944) and in the United States of America it was shown that school children between twelve and fourteen years of age, living in an area where the water contained 1·0 parts per million of fluorine experienced only about one-third as much dental caries as children in a fluorine-free area (Dean, 1949). Controlled experiments are now being carried out in America to determine whether a similar effect can be obtained from artificial fluoridation of water supplies, sodium fluoride being added to the water to bring its fluorine-content up to 1·0 to 1·2 parts per million.

From the few surveys carried out on adults in this country it would appear that the benefit which they derive from having been bred in a high-fluorine area is slight; it has been assessed as equivalent to a delay of five years in the onset of caries (Weaver, 1948 and 1950). Other opinions are that its effect is only transitory and that the adult population does not experience any benefit (Lancet 1951). In the United States of America, on the other hand, investigations have shown that the protection afforded by fluorine to the teeth of children is continued in adult life (Deatherage 1943 and McKay 1948): a reduction in caries incidence has been observed up to 45 years of age.

The present work was undertaken to obtain further information on the effect of the ingestion of fluorine on the dental condition of adults. At the same time, children below school age were included in the survey to see how early in life the effects of fluorine become apparent. This was a pilot investigation and the results indicate the association between dental caries and the fluorine content of the water. More precise information can come only from a larger study.

Selection of areas

Most of the drinking water in England contains from 0·0 to 0·25 parts per million of fluorine but in a few districts the public water supplies contain from 0·8 up to 2·0 parts per million and three small communities are known to have water supplies containing as much as 4·0 or 5·0 parts per million. The small size of these communities prevented their inclusion in this survey.

Town water supplies are frequently drawn from more than one source. Where the fluorine content of all sources is similar, such as from a trace to 0·25 parts per million, the multiplicity of supplies was considered immaterial in the present survey; but where a number of water supplies, of widely varying fluorine content, feed into the mains at different times such a district was considered unsuitable. In Chelmsford, for example, there are five sources of supply which vary in fluorine content from 0·0 to 6·0 parts per million and the amount of water drawn from each is not constant. The fluorine in such a supply might be sufficient to affect the caries incidence but the findings of a survey could not be assessed as the average fluorine content of the water could not be determined.

Three widely separated high-fluorine areas and three comparable low-fluorine areas were selected for examination. In this way a comparison could be made under conditions which might vary in different parts of the country and the effect on the caries incidence of factors other than fluorine could be minimised.

The high-fluorine areas selected were, South Shields in North East England, Colchester in East Anglia and Slough in the Thames Valley. The low-fluorine areas were North Shields, Ipswich and Reading. Each was in the same neighbourhood as its high-fluorine counterpart and resembled it closely except for the fluorine content of its water supply. North Shields was thus compared with South Shields, Ipswich with Colchester and Reading with Slough.

Fluorine content of the Water Supplies

Since the investigation was to include adults whose dental condition might have been affected by the fluorine content of the water during their childhood the histories of the water supplies were traced over the past forty years. It was found that there had been no significant change in the sources of supply but in most areas fluorine estimations were not available until recently.

Special analyses of the water supplies were made for the purpose of this investigation by the Department of the Government Chemist, numerous samples being taken in each area over a period of some months and at different times of the day. Two methods of analysis were used, a direct colorimetric method with all samples (Lamar, 1945, Maier, 1950) and a distillation method as a check in some areas (Willard and Winter, 1933, Hoskins and Ferris, 1936).

TABLE I

Average Fluorine Content of Water Supplies

	High Fluorine Areas.			Low Fluorine Areas		
	South Shields	Colchester	Slough	North Shields	Ipswich	Reading
Number of Samples	46	13	21	26	24	24
Average Fluorine content in points per million ...	0·82	1·45	0·9	0·07	0·3	0·1
Range	0·7–1·0	1·2–2·0	0·85–1·2	0·0–0·1	0·2–0·5	0·0–0·2

It was found that the amount of fluorine varied and Table I shows the average fluorine content for each area. Although it is not known what it may have been some 30 to 40 years ago it was presumed that little change had occurred as in each of the high-fluorine areas selected the teeth of some of the women in the higher age groups were slightly mottled.

Selection of Subjects

To obtain groups of like social status, mothers attending ante-natal and infant welfare centres and children attending day nurseries, infant welfare centres and nursery schools were examined. Only those who were born and had spent their childhood in the district were examined. In some towns it was found that only one of twenty mothers attending the clinics came into this category and at the most it was never more than one in five. In all, 268 mothers and 434 children were seen in high-fluorine areas and 296 mothers and 356 children in low-fluorine areas.

Method of Examination

The standard of dental examination was identical in all groups. Examinations were carried out by two of us (J. R. F. and G. P. J.), each examining half of every group. This was done to minimise personal differences of observation. Clinical findings were dictated by the observers to trained recorders who entered the details on special charts.

Clinic No. 6 probes were used, as their points were found to vary very little. Each probe was checked under a microscope before use and was sharpened and re-checked after being used four times.

Every tooth was classified as sound, carious, filled or missing. Missing third molars were counted as having been lost owing to caries unless it was clinically evident that they had not erupted. Missing deciduous lower incisors in four year old children were counted as naturally shed and not lost owing to caries.

The area of each cavity or filling was noted and the depth of the lesion was assessed. Four grades of depth were recorded:—(*a*) pits or grooves in the enamel diagnosed as carious by the clinical " sticking " of the probe; (*b*) deep pits where caries had penetrated into the dentine; (*c*) extensive caries invading the pulp; and (*d*) pulp involvement with periapical infection.

From such records several methods of caries assessment can be made. In this report decayed, missing and filled teeth are combined in the abbreviation D.M.F.

Examinations had to be carried out in centres and schools and it was not possible under these conditions to take bite-wing x-rays for interstitial caries, nor could the teeth be more than wiped dry with celotex. It is realised, therefore, that some interstitial cavities and early caries may have been overlooked but the method of examination was similar in all groups.

TABLE II

Incidence of Dental caries among Expectant and Nursing Mothers

Age	Number of mothers examined.		Average number of D.M.F. teeth per mother.		Reduction in high compared with low Fluorine Areas
	Fluorine Areas		Fluorine Areas		
	Low	High	Low	High	
					Per cent.
20 years and under...	22	17	12·5	8·5	33
21–25 years	92	91	16·2	10·0	38
26–30 years	107	69	19·3	12·5	35
31–35 years	40	61	21·5	16·2	25
36–40 years	30	23	22·8	19·2	16
Over 40 years ...	5	7	26·4	22·0	17

FINDINGS

Expectant and Nursing Mothers

Table II combines the data for mothers in the three high- and three low-fluorine areas and the separate data for the different towns are shown in Table III. Table II shows that at each age the caries incidence was lower in the high-fluorine areas than in the low-fluorine areas. It is to be noted, however, that the difference between the two types of areas decreases as the age of the mothers advances. Thus up to 30 years of age the difference is about 35 per cent. and at 36 to 40 years it has fallen to 16 per cent. The difference can also be expressed, as suggested by Weaver, as a delay in the onset of dental caries by a certain number of years. This value can be obtained by noting how many years it takes for the D.M.F. rate of a group in the high-fluorine areas to reach the same level as a group in the low-fluorine areas. It will be seen from Table II that throughout the age range examined there is a difference between the two groups of 10 years in the attainment of the same average D.M.F. value. For example, the average number of D.M.F. teeth per mother in the low-fluorine areas was 19·3 at 26 to 30 years; in the high-fluorine areas this incidence was not reached until 36 to 40 years. This suggests that mothers born in these high fluorine areas have the advantage of 10 years delay in caries experience.

TABLE III

Incidence of caries among Expectant and Nursing Mothers in the various High and Low Fluorine Areas

Average Number of D.M.F. Teeth per Mother

	Low Fluorine Areas						High Fluorine Areas					
	North Shields		Ipswich		Reading		South Shields		Colchester		Slough	
Fluorine content of water—p.p.m.	0·07		0·3		0·1		0·82		1·45		0·9	
Age	Number examined	D.M.F.	Number examined	D.M.F.	Number examined	D.M.F.	Number examined	D.M.F.	Number examined	D.M.F.	Number examined	D.M.F.
20 years and under ...	8	14·9	6	9·2	8	12·6	10	8·3	2	2·0	5	11·6
21–25 years	20	17·3	35	13·8	37	17·9	49	10·3	23	10·3	19	9·1
26–30 years	34	22·7	37	17·6	36	17·9	35	13·3	24	10·7	10	14·1
31–35 years	12	23·4	14	18·6	14	22·7	23	13·3	20	16·6	18	19·6
36–40 years	10	24·4	11	20·9	9	23·3	8	25·7	6	13·7	9	17·0
Over 40 years	2	30·0	—	—	3	24·0	1	25·0	4	21·5	2	22·0

Table III shows that in each of the three pairs of towns compared, North Shields and South Shields, Ipswich and Colchester, Reading and Slough, the caries incidence was less in the high- than in the low-fluorine areas. It will be observed that there was a noticeable variation in the caries incidence of the three low-fluorine areas, although the fluorine content only varied from 0·1 to 0·3 parts per million, and that in Ipswich the dental condition of the mothers was better than in either of the other low-fluorine areas.

The difference in the clinical condition of mothers of the same age in the two groups was more striking than the D.M.F. count would suggest. For example, among mothers aged 26 to 30 years those in the high-fluorine group had, on average, 6·7 missing teeth whereas the comparable figure in the low-fluorine group was 11·9. In addition, among mothers in the low-fluorine group caries was more severe and of the teeth which were present and decayed 12 times as many showed apical infection.

When mothers were grouped according to the amount of their D.M.F. experience it was found that at every age there were more women with a large number of D.M.F. teeth in the low- than in the high-fluorine areas. Thus among mothers aged 21 to 25 years, in the high-fluorine areas it was found that 17 per cent. had 4 or less D.M.F. teeth, 63 per cent. had 5 to 14 D.M.F. teeth and only 20 per cent. had 15 or more D.M.F. teeth. In comparison, none of the low-fluorine group had 4 or less D.M.F. teeth, 35 per cent. had 5 to 14 D.M.F. teeth and as many as 65 per cent. had more than 15 D.M.F. teeth. The reduction in caries incidence was apparent in all groups, which shows that the average D.M.F. figure had not been affected by a small number of individuals with a dental caries experience greatly above or below the average.

Among the 268 mothers examined in high-fluorine areas severely mottled or pigmented teeth were not found. In the few instances where mottling occurred it was so slight that it was detected only by careful clinical examination

Young Children

The percentage difference in caries incidence of children under school age between the combined high- and low-fluorine areas is shown in Table IV.

TABLE IV

Incidence of Caries among Young Children

Age	Number of children examined		Proportion of children caries free		Average D.M.F. teeth per child		Reduction in high compared with low Fluorine Areas
	Fluorine Areas		Fluorine Areas		Fluorine Areas		
	Low	High	Low	High	Low	High	
			Per cent.	Per cent.			Per cent.
2 years	55	23	26	61	2·9	1·2	59
3 years	138	166	15	40	4·4	2·2	50
4 years	163	244	16	27	4·6	3·1	33

The difference expressed as a delay is approximately two years. The data show that the reduction in dental caries observed in high-fluorine areas was already apparent to a considerable degree in the pre-school child. Not only was the number of immunes greater in the high-fluorine areas but the over-all caries experience was lower.

In Colchester which is the area with the highest concentration of fluorine in the drinking water it was found that the incidence of caries was lower than in any other area. The average D.M.F. per child was 0·0 at 2 years, 1·8 at 3 years and only 2·2 at 4 years.

TABLE V

Percentage of Children with varying numbers of D.M.F. Teeth

Age	Number of children Examined		Number of D.M.F. teeth					
			Less than 3		3 to 7		More than 7	
	Fluorine Areas		Fluorine Areas		Fluorine Areas		Fluorine Areas	
	Low	High	Low	High	Low	High	Low	High
			Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
2 years	55	23	57	83	36	13	7	4
3 years	138	166	35	60	46	36	19	4
4 years	163	245	35	53	47	35	18	12

Table V shows the percentage of children with varying numbers of D.M.F. teeth. It will be seen that in the high-fluorine areas there was a greater proportion with less than 3 D.M.F. teeth and that a greater proportion of children in low-fluorine areas had more than 3 D.M.F. teeth.

Discussion

This was a pilot survey restricted to small groups of mothers and children in three high- and three low-fluorine areas. The results are consistent and show a lower incidence of caries in the high-fluorine areas. It is important to note that both for mothers and children the percentage difference between the high- and low-fluorine areas decreases with age, but for children the lower caries incidence is equivalent to a delay of 2 years and for mothers to 10 years.

Not only is it important to obtain more precise information for the age groups studied, but also to establish the effect on older people. Moreover the present survey was restricted to women and children: it would be useful to know if the same differences could be observed among men . To obtain such information a more extensive study is necessary.

Summary

The dental caries experience of 268 adults in 3 high-fluorine areas was compared with that of 296 adults in 3 low-fluorine areas.

The age of the adults ranged from under 20 to over 40 years.

The fluorine content of the drinking water ranged from 0·82 to 1·45 parts per million in the high-fluorine areas and from 0·07 to 0·3 in the low fluorine areas.

It was found that in the high-fluorine areas:—

(*a*) the incidence of caries was lower in every age group. The reduction as compared with low-fluorine areas amounted to:—

20 years and under	33 per cent.
21–25 years	38 per cent.
26–30 years	35 per cent.
31–35 years	25 per cent.
36–40 years	16 per cent.
Over 40 years	17 per cent.

(*b*) there was apparently a delay of 10 years in the onset of caries.

(*c*) a smaller proportion of the D.M.F. teeth were actually missing and caries attack was less severe in the teeth which were present.

The dental caries experience of 434 children, aged 2–5 years, in 3 high-fluorine areas was compared with that of 356 children in 3 low-fluorine areas.

It was found that in the high-fluorine areas:—

(*a*) a reduction in dental caries incidence was already apparent at from 2–3 years of age. The reduction amounted to 59 per cent. at 2 years, 50 per cent. at 3 years and 33 per cent. at 4 years.

(*b*) the proportion of children who were free from caries was greater.

(*c*) there was a greater proportion with less than 3 D.M.F. teeth and a smaller proportion with more than 3 D.M.F. teeth.

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POLIOMYELITIS IN WALES DURING 1950

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During 1950, Wales shared with the rest of Great Britain a high incidence of poliomyelitis. Cases appeared early in the year so that it was expected to be a year of high incidence and so it proved, almost equalling the record high incidence of 1947.

When it was realised in 1949 that a large number of cases were appearing, it was decided to try to gain information of each case and so to build up a useful picture of poliomyelitis in Wales during the year. A questionnaire was forwarded to each medical officer of health and any point which was not satisfactorily dealt with, usually resulting from the fact that the patient was in hospital, was checked by contacting the hospital.

This procedure was followed again during the year 1950 and this paper deals with the analysis of the information obtained. Such a picture, of a disease so difficult to treat, both from the preventive and therapeutic points of view, may be of some value.

The number of patients involved may not correspond exactly with the Registrar-General's number of notifications as some patients were members of the Armed Services and when the information required for the questionnaire was not obtainable, these cases were not included. The number of these patients was few so that the picture was hardly altered by their omission and a reasonably accurate picture was maintained.

Incidence

The number of cases occurring in Wales during 1950 thus investigated was 380 and the latest available estimate of population by the Registrar General was 2,591,000.

Thus the incidence of the disease during the year was 14·7 cases per 100,000 living. This almost equals the rate of the record epidemic year, 1947, in this country when the rate was 15·0 cases per 100,000 living.

Such a repetition within three years and, with the history of 1938, 1947, 1949 and 1950, seems to show that the more fortunate position of the British Isles relative to some other countries before these years is disappearing.

Geographical Areas

The terms " urban " and " rural " does not necessarily indicate the type of authority as some previously rural areas have become urbanised, a few almost wholly and others in part. However, this has not happened to such a degree as to make a glance at the position not worthwhile.

	Urban Areas	*Rural Areas*
1947	14·0	17·0 per 100,000 living
1949	5·3	3·6 ,, ,, ,,
1950	12·2	19·3 ,, ,, ,,

The number of patients in 1950 from urban areas was 230 and from rural areas 150, giving the rates shown above. In 1947 and 1950, years of high incidence, the rural areas rate surpassed that of the urban areas but, allowing for the urbanisation of some, it is thought that there is really little difference.

1950

Local Health Authority	*Number of Cases*	*Rate per 100,000 Living*	*Local Health Authority*	*Number of Cases*	*Rate per 100,000 Living*
Anglesey	4	8	Monmouthshire ...	19	5·9
Breconshire	6	10·7	Montgomeryshire ...	20	43·7
Caernarvonshire ...	25	22	Pembrokeshire ...	11	12·3
Cardiganshire	3	5·5	Radnorshire	6	29·6
Carmenthenshire ...	32	18·7	Cardiff...	16	6·6
Denbighshire	43	25·5	Merthyr	—	—
Flintshire	20	13·8	Newport	8	7·5
Glamorgan	107	14·6	Swansea	54	33·7
Merioneth	6	14·7			
			Total ...	380	14·7

No clear findings result from comparing rates in 1949 and 1950 which would indicate that a high incidence during one period might, as a result of community resistance which developed, tend to have a low incidence during the following visitation. This will be seen from the table below.

Areas of High Incidence		*Areas of Low Incidence*	
1949	1950	1949	1950
Swansea	Montgomeryshire	Caernarvonshire	Merthyr
Cardiff	Swansea	Denbighshire	Cardiganshire
Glamorgan	Radnorshire	Merioneth	Monmouthshire
Flintshire	Denbighshire	Radnorshire	Cardiff
Monmouthshire	Caernarvonshire	Merthyr	Newport

Two of the authorities in the 1949 high incidence list, however, appear in the low incidence list in 1950 and three in the low incidence list in 1949 appear in the high incidence list of 1950.

Distribution by month

Month	*Number of Cases* 1949	1950
January	—	7
February	—	6
March	2	5
April	4	2
May	1	8
June	7	14
July	25	50
August	27	103
September	26	76
October	35	68
November	22	31
December	12	10

The increase in the number of cases began at least a month earlier in 1950 than in 1949, suggesting the probability of a high incidence which in fact occurred. The peak month in 1950 was August, whereas in 1949 was delayed until October.

Sex Distribution

Males – 223 or 59% of all cases
Females – 157 or 41% of all cases

These proportions closely resemble those in 1947 when the males accounted for 57% of the total.

Ages of Patients

Incidence in Age Groups

Age	*Number of Patients*	*Per cent. of total*		
0–1	15	3·9	40·3	70·5
1–2	36	9·4		
2–3	45	11·8		
3–4	38	10·0		
4–5	20	5·2		
5–10	75	19·7		
10–15	40	10·5		
15–20	30	7·9	29·1	
20–30	48	12·8		
30–40	22	5·7		
40–50	5	1·3		
50–60	2	0·5		
60–70	1	0·2		
Not ascertained	3	0·7		

The comparison between 1949 and 1950 is as follows:—

Age	1949 per cent.	1950 per cent.
Under 5 years	58	40
Under 15 years	81	71
Over 15 years	19	29

Thus " infantile " paralysis occurred in 29 per cent. of cases in persons over 15 years of age and in 21 per cent. of cases in persons over 20 years of age. The 1950 distribution shows a distinct trend inasmuch as the incidence increased in the older groups and decreased in the younger age groups. But, in spite of these changes in age distribution, the point of highest incidence in both years still remained the same, namely, the 0 to 5 years group.

Housing Conditions

Overcrowding

In 22 instances out of 380 cases there was some evidence of overcrowding.

No. of persons in house	*No. of households*
1	1
2	10
3	53
4	110
5	77
6	57
7	19
8	19
9	10
10	2
11	4
12	—
13	1
14	1
15	1
School or Nursery	2
Camp or Hotel	9
Not known	4

Sanitation

In only 21 instances was there any indication of any serious or even moderately bad sanitary conditions and the sanitary conditions of the houses did not seem to play any substantial role in the incidence or spread of the disease.

Source of Infection

Recent Family Illness

To discover possible contact with a missed case the question was asked whether there had been recent personal or family illness. In 21 instances illnesses were noted but, in most, the nature of the illnesses was not significant. This does not mean that one could be satisfied that missed cases were not occurring but rather that no real search had been made.

Contact with known cases of poliomyelitis

In 31 instances out of 380 notified cases of anterior poliomyelitis for which returns were made, there was evidence of recent contact with a patient suffering from the disease.

More than one notification in same household

There were eight instances in which there was more than one case notified from the same household. In these eight households there were in all 45 people at risk, distributed as follows: 5, 6, 4, 4, 4, 8, 8 and 6. In three of these instances there were three patients involved. Not more than six days appeared to separate any of the cases occurring in the same household as determined by the onset of the earliest symptoms.

Possible " Precipitating " factors

Fatigue

In 26 instances there was evidence of enough physical effort to cause fatigue. Such forms of exercise leading to fatigue included cycle tours, swimming, climbing, work of the blacksmith, a confinement, work of the postman in a rural area, farm labouring under exceptional circumstances, travelling excessive distances without resting at nights, playing in the snow, and playing in a football match.

Attention was directed in these instances to whether the particular form of fatigue could be related to the location of the paralysis, but no reliable evidence of this relationship was found.

One feature of importance, and this was that in these 26 cases in which there was evidence of fatigue, there were six deaths, a case fatality of 28 per cent., whereas the total cases, numbering 380, produced only 21 deaths, a case fatality of 5·5 per cent.

Recent Operations

In 4 instances there was a history of recent surgical operations and there was nothing of particular consequence in these cases. There were no instances of recent tonsillectomy.

Relation to Diphtheria Immunisation

In relation to nine patients, there was a history of recent immunisation against diphtheria.

Case 22 Child aged $1\frac{4}{12}$ years.
Antigen—Combined diphtheria and whooping cough.
Last injection given about 6 weeks before symptoms.
Injection into thigh but paralysis of left upper arm developed.

Case 47 Child 2½ years.
Antigen—Diphtheria.
Last injection nearly two weeks before onset of disease.
Injection into left arm. Paralysis of right shoulder muscles developed.

Case 79 Child aged 1 year.
Antigen—Combined diphtheria and whooping cough.
Last injection given 4 weeks before onset of disease.
Injections into left arm and left buttock—paralysis occurred in right shoulder muscles and left leg.

Case 83 Child 5 years.
Antigen—Diphtheria (A.P.T.).
Last injection three weeks before first symptoms of disease.
Injection into left arm—Paralysis occurred in left arm.

Case 98 Child aged $1\frac{1}{12}$ years.
Antigen—Diphtheria and whooping cough.
Last injection given four weeks before onset of disease.
Injection into left leg and paralysis occurred in left leg.

Case 128 Child aged $1\frac{8}{12}$ years.
Antigen—Diphtheria.
Last injection—3 weeks before onset of disease.
Injection into left arm. Patient suffered severe paralysis of legs.

Case 133 Man 18½ years.
Schick Test and vaccination against smallpox.
Last injection 3 weeks before onset of disease.
Injection etc. left arm, no paralysis occurred.

Case 198 Child aged $1\frac{1}{12}$ years.
Antigen—Combined diphtheria and whooping cough.
Last injection about 7 weeks before onset.
This given into right thigh and patient had paralysis of the right leg.

Case 328 Child aged 9 months.
Antigen—Diphtheria.
Last injection 11 days before onset of symptoms.
Injection given into left upper arm—developed paralysis of left arm.

There seemed to be no common pattern as far as the lapse of time after injection before onset of first symptoms of the disease or any special relationship between the site of the injection and subsequent paralysis. However, in four patients the paralysis occurred in the limb in which the antigen had been injected.

Recent Trauma

It has been suggested that trauma to a particular part may determine the site of paralysis in some cases. No decision has been possible. Some examples, but too few and indefinite to be in any way conclusive, are given in order to show that further observation is needed.

Case 136 A soldier, 21 years old, was vaccinated against smallpox and also received T.A.B. inoculation in the left upper arm on the 22nd June, 1950. Anti-tetanic toxoid in the right upper arm was given around this time. A second T.A.B. inoculation was given on the 15th July, 1950.

On the 9th August the patient was taken ill with anterior poliomyelitis, developing a paresis of the left upper limb.

Case 336 A girl, fifteen months old, injured her left thumb by catching it in a door. Two weeks later she became ill with poliomyelitis and developed paralysis of some of the muscles of her left forearm.

Case 351 A man, aged forty-seven years cut a finger of his right hand. Two weeks later he became ill with the disease and developed a paralysis of his right arm.

Case 355 A boy, one and a half years old fell in his bedroom on the 23rd November, 1950, hurting his left leg. On the 30th November he was admitted to an Isolation Hospital with anterior poliomyelitis showing evidence of a slight weakness of muscles of his left foot and calf.

Earliest symptoms noted

Earliest symptoms or signs noted	*Number of occasions*	*Earliest symptoms or signs noted*	*Number of occasions*
General Malaise	31	Nausea or Vomiting ...	28
Headache	78	Sore throat	17
Fretfulness	19	Paralysis	15
Convulsions	1	Stiff neck	6
Pain in Limb or Part ...	38	Bronchitis	1
Tiredness	20	Cold, etc.	23
Drowsiness	8	Abdominal pain	4
Fever	39	Doubtful	52

The most common symptom at the onset of illness, in order of frequency, was headache, pain in limb or part general malaise, nausea or vomiting, and symptoms of feverish cold—

Degree of Paralysis

An analysis of the replies to this question showed this as follows:—

Degree of Paralysis	Number of Patients	Per cent. of Total (approx.)	
		1950	1949
Severe	69	18	15
Moderate	109	29	27
Slight	87	23	24
Transient	10	3	6
None	103	27	8
Not known	2	0·5	20

It must be remembered that the allocation to these categories was made soon after the onset of the disease and might have varied substantially from a review taken some months later.

In 1950, 27 per cent. of the cases were non-paralytic.

Site of Paralysis.

Site of Paralysis	Number of Cases	Per cent. of Total
Face muscles	21	5·5
Arm	48	12·6
Leg	119	31·3
Limb plus other muscles	45	11·8
Arm and leg	24	6·3
Neck muscles	6	1·6
Respiratory muscles	7	1·8
Palate	1	0·3
Back muscles	4	1·0
No paralysis	103	27·0
Not known	2	0·5
Total	380	100·0

A lower limb was involved in about 39 per cent. of all cases and this is almost the same as in 1949.

Polioencephalitis and Poliomyelitis

Out of 380 patients, 361 were diagnosed as having poliomyelitis and 19 as polioencephalitis, i.e. in 5 per cent. of cases. This is about half the polioencephalitis percentage of the previous year. Ten of these 19 patients died.

Deaths

Twenty-one patients died, a case fatality rate of 5·5 per cent. and a death rate of 8·1 deaths per million living. The comparable figure for the latter rate for 1949 was 2·7.

Summary

(1) The incidence of poliomyelitis in Wales in the year 1950 was almost equal to the record high incidence year of 1947. The incidence was 14·7 per 100,000 living.

(2) As in 1947 the rate of incidence was greater in rural than in urban areas

(3) Males were attacked more often than females, i.e. in 59 per cent. of cases This too, followed the experience of 1947.

(4) About 29 per cent. of cases were in persons over 15 years of age but the highest incidence fell on the age group—0 to 5 years. Compared with 1949, the proportion of older people attacked was larger.

(5) There was little evidence of overcrowding or indifferent sanitary conditions being implicated in the causation or spread of the disease.

(6) In only eight instances out of 380 cases was there evidence of more than one case occurring in the same household.

(7) Fatigue was mentioned in twenty-six instances, but six of these patients died.

(8) Nine of the 380 patients had been recently immunised against diphtheria or against diphtheria and whooping cough.

Cases are given where local trauma may have been a pre-disposing cause of determining the site of paralysis, and it is thought this relationship requires wider observation.

(9) The earliest symptoms of the disease in order of frequency were headache, pain in a limb or part eventually affected, general malaise, nausea or vomiting and feverish cold. In 1949 the same order was found.

(10) In 39 per cent. of all cases a lower limb was involved.

(11) The degree of paralysis was described as severe in 18 per cent., moderate in 29 per cent. and slight in 23 per cent. of cases. 27 per cent. of all cases were non-paralytic.

(12) 5 per cent. of all cases were diagnosed as polioencephalitis but these provided 48 per cent. of the deaths.

(13) Twenty-one deaths occurred in 380 patients, a death rate of 8·1 per million living. The case fatality of the disease was 5·5 per cent.

I wish to express my gratitude to all those who so kindly returned questionnaires, assisted by giving further information, or helped in any other way.

NOTIFICATION OF INFECTIOUS DISEASES IN ENGLAND AND WALES, APRIL, 1951

Issued from the General Register Office, Somerset House, W.C.2.

	April 7th	April 14th	April 21st	April 28th	Average weekly figures for April 1950
Scarlet Fever	845	823	948	1,084	1,149
Whooping Cough	4,010	3,838	3,498	3,648	2,633
Diphtheria	48	42	32	32	60
Measles, excluding Rubella ...	28,829	22,656	19,025	18,036	6,593
Acute Pneumonia	799	707	551	571	715
Meningococcal Infection ...	39	46	40	29	31
Acute Poliomyelitis (Paralytic) ...	11	15	14	11	17
Acute Poliomyelitis (Non-paralytic)	4	5	8	8	5
Ophthalmia Neonatorum ...	34	44	41	45	45
Puerperal Pyrexia and Puerperal Sepsis	68	77	61	85	80
Dysentery	846	858	1,002	873	338
Paratyphoid	27	13	12	4	3
Typhoid	8	6	4	4	2
Smallpox	—	—	—	—	—

No cases of Cholera or Plague.
1 case of Typhus Fever (not louse borne) in week ending 14th April.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Reading Laboratory : Change of Address

The address is now: Public Health Laboratory, Battle Hospital, Reading (*Tel.:* Reading 2957). Dr. Norman Wood remains in charge of the laboratory.

Birmingham Laboratory : Change of telephone number

The telephone number is now: Central 6921/2.

Correction

March, 1951, *number.*—In List C on p. 70, the telephone number of the laboratory at Burnley is wrongly given as Burnley 3237; it is in fact 3714.

THE EFFECT OF PASTEURIZATION ON MILK CONTAINING RICKETTSIA BURNETI

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Q fever, an acute febrile illness with pneumonia caused by *Rickettsia burneti*, was discovered in Queensland, Australia, by Derrick (1937). Epidemiological studies there suggested that occupational contact with cattle was responsible for infection. The mode of infection was believed to be the inhalation of dust containing infective tick excreta deposited on the hides of the cattle. Later, large outbreaks of the disease in troops in the Mediterranean theatre of war again appeared to be dust-borne in origin. In 1947, Q fever was found to be endemic in Southern California and dust-borne infection from farm animals was found to account for some of the cases.

The discovery by Huebner *et al.* (1948) that raw milk from a number of dairy herds in the Los Angeles area contained *R. burneti* provided an additional explanation to the inhalation of contaminated dust as the mode of infection. Preliminary investigations of 300 cases of Q fever in Los Angeles (Beck *et al.* 1949) showed that about a third of them might be accounted for by the consumption of raw milk; and later a systematic serological survey by Bell, Beck and Huebner (1950) reinforced this finding when they found that the incidence of positive complement-fixation tests against *R. burneti* was about ten times more frequent in persons drinking raw milk than in their neighbours who did not consume it.

Q fever in cattle appears at present to be a benign and possibly symptomless disease and its epidemiology is obscure. For these reasons, it is difficult to devise any method of controlling it, and pasteurization of infected milk would seem to be the most logical method of protecting human beings against milk-borne infection.

Rickettsia burneti is reputedly rather heat-resistant and for this reason Huebner *et al.* (1949) investigated the effect of pasteurization on naturally infected milk. Commercial plants of the high-temperature short-time (H.T.S.T.) and the vat-holding types were tested using bulked milk from two herds known to be infected. Exposure to a temperature of 160·5°F. for 15 seconds was successful in eliminating *R. burneti* from the milk in each of four experiments with the H.T.S.T. method. However, in the vat-holding method, using a temperature of 143°F. for 30 minutes, milk from one of four trials was found still to contain *R. burneti*. Pasteurization by the "holder" method, using the time-temperature combination required by Californian law, was not uniformly successful, therefore, in making the milk non-infective. The same workers also found that 2 of 31 samples of commercially pasteurized milk with negative phosphatase reactions were still infective for guinea-pigs. The importance of using an efficient pasteurization method to remove *R. burneti* from milk has led us to reinvestigate the H.T.S.T. method and to carry out a small number of laboratory experiments on the "holding" method. Milk from an infected herd and milk to which infected yolk sac had been added was exposed for 15 seconds at various temperatures ranging from 154°F. to 163°F. in a small H.T.S.T. machine. In the laboratory experiments, tubes containing milk with added infected yolk sac were held for 30 minutes in a water-bath at various temperatures ranging from 132°F. to 149°F. The presence of *R. burneti* in heated and unheated milks was determined by guinea-pig inoculation.

Materials and methods

Milk. Known infected milk was obtained from a farm (Farm W., *see* Marmion and Stoker, 1950) in N.E. Kent. The milk was purchased by permission of the Milk Marketing Board and brought by road to the pasteurizing plant in south London. The pooled morning and evening meals, which had been cooled and held at room temperature, amounted to about 30 gallons and were transported in three 10-gallon cans; on arrival these were emptied into three other cans so that the whole was uniformly mixed. The herd milk from Farm W had been positive on several occasions in the past, but to guard against a sudden reversion to the uninfected state during the experiment smaller quantities of milk were collected a month previously from two infected cows ("Ni" and "Eliz") in the herd and frozen at -15°C. after guinea-pig inoculations. In two of the experimental runs, this milk was thawed and added to the usual daily 30 gallons. The number of rickettsiae in the milk was increased in three experiments by the addition of infected yolk sac suspension to the naturally infected milk. On two occasions, the yolk sacs came from eggs infected respectively with the "Christie" and "M_1" strains of *R. burneti* isolated in Great Britain (Stoker, 1950), and on the third occasion yolk sacs infected with the "Henzerling" (Italian) strain were used. The yolk sac was in the form of a fine suspension prepared in the Waring Blendor and this was mixed with 600 ml. of the milk in a screw-cap bottle containing glass beads and shaken vigorously by hand. Equal portions were then added to each of three cans of the previously mixed milk and agitated well with a stirrer. We are indebted to Dr. M. G. P. Stoker of the Department of Pathology, Cambridge, for the yolk sac suspensions of the three strains of *R. burneti*.

Description of the H.T.S.T. plant and method of operation

In the H.T.S.T. method of pasteurization milk flows continuously over one side of a series of thin metal plates which are heated by the circulation of hot water on the other surface. The temperature of the milk rises rapidly and the flow rate of the milk, the surface area of the plates and the temperature of the hot water are all so adjusted that when the milk has just attained the correct pasteurizing temperature it leaves the heating section and enters a tube, called the "holding" tube, before passing to the cooling section. The milk takes about 15 seconds to pass through this tube and thus, apart from a slight loss of heat to the air, is "held" (i.e. maintained) for this period of time (the holding time) at the temperature it attained at the end of the heating section. At the end of the "holding" section the milk is rapidly cooled by passing over metal plates cooled on the other surface by cold water, or, in the heat-exchange section, by the incoming stream of unheated and unpasteurized milk. The total thermal effect on the milk is, therefore, the sum of the effects during heating, holding and cooling.

In the present investigation, an A.P.V. Company's stainless steel plate-pasteurizing plant, type H.T.C.a., modified for the experiment and arranged for treating milk at 50 gallons per hour (g.p.h.), was used. It consisted of a regenerative (heat-exchange) section, a heating section, a holding section and two water-cooling sections.

The temperature of the hot water used to heat the milk to its final temperature was maintained automatically by an air-relay controller. The setting of this controller could be altered at will; it was found in practice that the milk temperature at a point near the beginning of the holding tube could be adjusted from one set value to the next lowest required in about one minute.

A mercury-in-glass thermometer with its bulb inserted in the milk flow at the beginning of the holding tube was used to determine the temperature of the milk. This thermometer had been previously compared with a thermometer certified by the National Physical Laboratory and found to be accurate to within 0·1°F. over the range of temperatures used in this experiment. During experimental runs the temperature of the milk at the beginning of the holding tube was observed every 10 seconds and recorded every 30 seconds. Comparisons of the hot water and milk temperatures facilitated downward adjustment of the milk temperature during experimental runs.

The holding section consisted of a tube of 1½ inch outside diameter and was of a length calculated to give a holding time of 15 seconds with a flow rate of 50 g.p.h. The actual holding time was checked using the nickel glyoxime method of Dummett and Mongar (1944) with hot water circulating instead of milk. The length of that portion of the holding tube between the point of injection of the nickel chloride and the point of sampling was only 43·5 inches so that for a total holding time of 15 sec. the nickel chloride would be detected at the sampling point in $\frac{43\cdot5}{60\cdot5} \times 15 = 10\cdot8$ seconds after injection. Accordingly, the flow rate was adjusted to 48 g.p.h. when negative results were found with the Dummett and Mongar test at 10 seconds but a positive result at 11 seconds. The velocity of flow of the milk through the holding tube differs in various parts of the stream and consequently some particles of milk (or nickel chloride) traverse it either quicker or slower than the average particle, which should take 15 seconds. If a comparison is made of the actual time taken for the first detectable nickel chloride to pass through the test section of the tube with the calculated time assuming the whole cross section of liquid in the tube to be moving at uniform velocity, an estimate, the " holding efficiency " of the system, can be made of the ratio of the time of traverse of the fastest particles and the average time taken by all particles. In the plant used the holding efficiency was calculated to be 70 per cent.

Throughout each experimental run the flow rate was kept constant at 48 g.p.h. ± 2 per cent. by hand adjustment of a rate-cock to give a constant forward pressure in the system. The flow rate of water at the set pressure through the plant when working normally was checked at the beginning of each daily experimental run.

A total of approximately 30 gallons of infected milk was available for each day's experiments and it was planned to treat this milk at the following temperatures:—

163–163·5°F: 162–162·5°F: 161–161·5°F: 160–160·5°F:
159–159·5°F: 158–158·5°F: 156–156·5°F: 154–154·5°F:

Two experiments were done with infected milk without any addition, two with infected milk plus stored milk from the two cows, and three with infected milk and added yolk sac suspension.

Each day's run was started at 163–163·5°F. and then the temperature was lowered successively till the whole series of treatments had been completed. The time taken for milk to flow through the entire plant, from the tipping tank to the sampling point at the outlet from the second water-cooling section, was found by the use of dye to be just over three minutes. The flow time from the entrance to the holding tube to the outflow was just under two minutes. Accordingly, the plant was operated at each of the specified temperatures, as observed on the thermometer at the beginning of the holding tube, for exactly four minutes before taking a sample of the heat-treated milk. At the end of each day's run, the plant was sterilized by circulating water at 180°F. for 10 minutes and cleaned without dismantling by the use of phosphoric acid followed by alkaline detergent and subsequent rinsing. Immediately before use the plant was again sterilized by circulating water at 180°F. for 10 minutes. All treated milk and milk residues and any rinse water likely to be infective were collected in a 100-gallon covered tank at the outlet of the machine. The contents of this tank were boiled by steam injection before being discharged.

Collection and testing of samples in the H.T.S.T. experiment

Samples of milk were collected into 25 ml. universal containers with rubber-lined screw caps. Unheated milk was taken from the tipping tank and heated milk collected as it emerged from the outlet pipe of the machine, and then the cap of the container was screwed home tightly and the whole bottle immersed in 1/2000 Chloros. The containers were then placed on solid carbon dioxide in a large vacuum flask and subsequently stored in an electric refrigerator at −60°C. To test for the presence of *R. burneti*, 3 ml. of each milk sample were inoculated intraperitoneally into two previously bled guinea-pigs (wt. 300–500 gm). These animals were caged with an uninoculated control guinea-pig, or in some instances a small number of control guinea-pigs were distributed at random among the cages. Guinea-pig cages were housed in small cubicles with ultra-violet light air sterilizer units. Guinea-pigs inoculated with unheated milks were housed in separate cubicles from those inoculated with heated milks. One month after inoculation, the guinea-pigs were killed and bled from the heart and a portion of liver and spleen collected from each guinea-pig. The pre-inoculation and post-inoculation samples of sera were tested for complement-fixing antibodies to *R. burneti*. Dilutions of the post-inoculation sera were at first " screened " at 1/10 and then positive samples and the appropriate pre-inoculation specimen were titrated to their end points. The sera were inactivated at 58°C. for 30 minutes, and the complement-fixation test was carried out using a calcium-magnesium saline throughout; $2\frac{1}{2}$ minimal haemolytic doses of complement, an optimal dose of antigen (yolk sac suspension of the Henzerling strain of *R. burneti*) and the serum were mixed in unit volumes of $0 \cdot 1$ ml. and held at 4°C. for 18 hours to allow fixation of complement. A sensitized cell system of $2\frac{1}{2}$ per cent. sheep red cells and 5 M.H.D. of haemolysin (final concentration) were then added and incubation was carried out for 30 minutes at 37°C. Controls for complement strength, and for un-inactivated complement in the guinea-pig sera after heating, were included. Dilutions of each positive serum were also tested against that dilution of a typhus vaccine (Connaught Laboratories) which was just not anticomplementary when tested alone so as to ensure that the fixation observed with the Q fever antigen was specific and not due to a summated anticomplementary effect of serum, yolk sac, and rickettsial suspension. A rise in titre from the pre-inoculation specimen at $< 1/5$ to 1/10 or 1/20 in the post-inoculation specimen is recorded in the tables as a doubtful positive (±) result. A rise from $< 1/5$ to 1/40 or over is designated positive (+). These serological criteria and their relation to infection of the guinea-pig are considered in the discussion.

Retesting of milk samples

When the result of the first test of the milk specimens was known, certain selected ones belonging to temperatures where the inactivation of *R. burneti* was equivocal were retested using a larger number of guinea-pigs for each sample, and in some instances a larger volume (4 ml.) of milk was injected into each guinea-pig. In this way, it was hoped to obtain positive results with milks containing only a few rickettsiae when the factor of "random sampling" would seriously affect the result. An attempt was also made to demonstrate *R. burneti* in the tissues of guinea-pigs with "doubtful positive" serological reactions by passage of liver and spleen suspensions to other guinea-pigs. This was uniformly unsuccessful.

The absence of any positive serological result in any of the uninoculated control guinea-pigs suggests that cross-infection did not occur and that the development of antibody was the result of the inoculum received by the animals.

Titration of unheated milks

Tenfold dilutions of milks were made in 10 per cent. broth saline using 9 ml. of diluent and 1 ml. of milk; 3 ml. of each dilution were then inoculated intraperitoneally into each of two guinea-pigs.

Protection of workers and disinfection of plant and premises in the H.T.S.T. experiment

All workers had received three 1 ml. doses of Q fever vaccine (Rocky Mountain Laboratory) kindly supplied by the U.S. Public Health Service. During the experiments, rubber gumboots, rubber gowns, rubber gloves, dust respirators and eye shields (light anti-gas type) were worn. Floors and wooden surfaces were repeatedly swabbed with lysol. Metal surfaces (parts of the plant, insides of cans, etc.) were sterilized with live steam. There were no clinical cases of Q fever among the operators. The need for these measures was subsequently justified by the recovery of *R. burneti* from air-sampler plates collected during certain manipulations such as mixing milk and yolk sac suspension.

Laboratory pasteurization of milk

A gas-heated water-bath, controlled by a bi-metallic strip thermostat and fitted with a "propeller type" electric stirrer, was used for this procedure. Raw milk was mixed with stored unheated milk containing "Christie" and "M_1" yolk sac suspension from the main H.T.S.T. experiment. Ten ml. quantities of this milk were pipetted into 6 × ½ inch test-tubes and any milk adhering to the walls of the top third of the test-tube was charred by flaming the glass. The tubes were sealed with "Astell" rubber seals, and for each temperature test two were placed in the water-bath in such a way that the level of the water on the outside of the tube was 2 inches above the level of the milk in the test-tube. The temperature of the milk in the water-bath was determined by a N.P.L. thermometer suspended in a test-tube containing uninfected raw milk. This tube was placed in the bath at the same time as the test batches of milk and the stop clock started for the 30-minute period when the temperature was within 0·5°C. of the desired test temperature. The temperature was recorded every 2 minutes during the 30-minute period and at the end of the period the milk tubes were rapidly cooled with running water. The presence of *R. burneti* in the milk was determined as in the H.T.S.T. experiment.

Phosphatase estimations

These were carried out on milk which had been stored at –60°C. for varying periods. Phosphatase was estimated by the standard Kay and Graham technique (Statutory Instrument No. 1589, 1949) except that milks were stored at –60°C. for periods up to two months before testing. To determine whether this storage at –60°C. had any deleterious effect, the following tests were made. A small quantity of raw milk was added to pasteurized phosphatase-negative milk to give a reading of 3·0 Lovibond blue units (L.B.U.) in the test. In addition, three milks incorrectly pasteurized by commercial plants, with readings of 3–4 L.B.U., were obtained and portions of each of the four milks stored at –60°C. At intervals, two observers, one with considerable experience of routine phosphatase estimations, tested samples removed from the –60°C. refrigerator. No significant difference was detected between the phosphatase values obtained with the original unfrozen milks and with those stored at –60°C. for periods up to four months.

Results

Table I shows the results obtained in seven experiments with the H.T.S.T. plant. It is evident that a temperature of 154–156°F. for 15 seconds was sufficient to eliminate *R. burneti* from the naturally infected milk (Exps. A, B, C and D) and from naturally infected milk to which the Henzerling strain of *R. burneti* had been added (Exp. G). In the two experiments, E and F, in which larger numbers of rickettsiae were present in the milk (see Table II for titres of rickettsiae in the untreated milk in each group of experiments), guinea-pigs were consistently infected with milk that had been heated to 158–159°F., and in repeat experiments (column 6, Table I) doubtful infection was obtained as high as 160–161°F. In these experiments (E and F) milk was also found to be still infective for guinea-pigs when the phosphatase test was negative. The significance of this finding and of the temperature at which the phosphatase became negative is considered in the discussion.

Laboratory pasteurization experiments

The results of these experiments are shown in Table III. In the first experiment in which milk containing about $1/3 \times 10^4$ guinea-pig infective doses of *R. burneti* per ml. (Table II) was exposed to a temperature of 143·5–148°F. for 30 minutes, the organisms were destroyed, but after exposure to a temperature of 142·7°F. the milk was still infective for guinea-pigs. In the second experiment, milk containing fewer rickettsiae ($1/3 \times 10^3$ guinea-pig infective doses per ml.) was not infective after heating at 141·9°F. for 30 minutes.

Discussion

A rising titre of complement-fixing antibodies to *R. burneti* in an injected guinea-pig is generally considered to indicate the presence of infective organisms in the inoculum. However, in the experiments E and F described above, the number of rickettsiae in the milk was rather large—about 10^5 guinea-pig infective doses per ml.—and it might be argued that the serological response observed in the guinea-pigs was the result of immunization with heat-killed rickettsiae rather than of infection with some that had survived the heating. There is no doubt that if guinea-pigs are inoculated with killed and concentrated suspensions of *R. burneti* some will develop a titre of complement-fixing antibodies of the same order as that observed in guinea-pigs convalescent from infection with *R. burneti* (Smadel *et al.*, 1948). Stoker (1951), however, did not find a significant titre of complement-fixing antibodies in guinea-pigs which had received phenolized yolk sac suspensions containing originally 10^5 or 10^6 guinea-pig infective doses of *R. burneti*, and we have found that guinea-pigs inoculated with 3 ml. of formolized yolk sac suspension containing originally 10^5 guinea-pig infective doses per ml. did not develop complement-fixing antibodies above a titre of 1/10 when the guinea-pigs were bled one month after inoculation. Consequently, it may be concluded that guinea-pigs with a titre of 1/40 or over, the designation + of the tables, had experienced infection rather than immunization with killed *R. burneti*. The distinction between infection and immunization by the conventional method of further passage of supposedly infected guinea-pig blood or tissue offers great difficulty when the original inoculum contains few viable rickettsiae as the guinea-pig does not develop a temperature and the optimal time for obtaining passage material is indeterminate ; consequently, little significance can be attached to a negative passage result.

TABLE I

The effect of treating milk infected with Rickettsia burneti at various temperatures for 15 seconds in a H.T.S.T. plant

Experiment	Type of milk	Treatment Temperature °F.	Phosphatase (Lovibond Blue Units)	Results of guinea-pig inoculation					
				First test Guinea-pig		Second test Guinea-pig			
				1	2	1	2	3	4
A	Naturally infected milk	Unheated♀	Strong positive	+	+	NT	NT	NT	NT
		163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	,,	—	—	NT	NT	NT	NT
		161–161·5	,,	—	—	NT	NT	NT	NT
		160–160·5	,,	—	—	NT	NT	NT	NT
		159–159·5	,,	—	—	NT	NT	NT	NT
		158–158·5	,,	—	—	NT	NT	NT	NT
		156–156·5	1·8	—	—	—	—	—	—
B	Naturally infected milk	163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	,,	—	—	NT	NT	NT	NT
		161–161·5	1·8	—	—	NT	NT	NT	NT
		160–160·5	<1·5	—	—	NT	NT	NT	NT
		159–159·5	1·5	—	—	NT	NT	NT	NT
		158–158·5	<1·5	—	—	NT	NT	NT	NT
		156–156·5	>1·8<2·1	—	—	—	—	—	—
C	Naturally infected milk+ stored milk ‘Ni’ and ‘Eliz’	163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	2·1—2·5★	—	—	NT	NT	NT	NT
		161–161·5	<1·5	—	—	NT	NT	NT	NT
		160–160·5	,,	—	—	NT	NT	NT	NT
		159–159·5	,,	—	—	NT	NT	NT	NT
		158–158·5	,,	—	—	NT	NT	NT	NT
		156–156·5	2·1	—	—	NT	NT	NT	NT
		154–154·5	>3<4	—	—	—	—	—	—
D	Naturally infected milk+ stored milk ‘Ni’ and ‘Eliz’	163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	1·5	—	—	NT	NT	NT	NT
		161–161·5	1·5	—	—	NT	NT	NT	NT
		160–160·5	1·5	—	—	NT	NT	NT	NT
		159–159·5	<1·5	—	—	NT	NT	NT	NT
		158–158·5	<1·5	—	—	NT	NT	NT	NT
		156–156·5	>1·5<1·8	—	—	NT	NT	NT	NT
		154–154·5	3	—	—	±	—	—	—
E	Naturally infected milk+ ‘Christie’ and ‘M_1’ yolk sac suspension	163–163·5	1·5	—	—	NT	NT	NT	NT
		162–162·5	<1·5	—	—	—	—	—	—
		161–161·5	,,	*—	*—	±	±	±	NT
		160–160·5	,,	*—	*—	±	±	±	NT
		159–159·5	,,	*+	*—	+	+	±	NT
		158–158·5	,,	+	—	+	+	±	NT
		156–156·5	3	+	+	NT	NT	NT	NT
		154–154·5	NT	+	+	NT	NT	NT	NT
F	Naturally infected milk+ ‘Christie’ and ‘M_1’ yolk sac suspension	163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	,,	*—	*—	—	—	—	—
		161–161·5	,,	*—	*—	±	—	—	—
		160–160·5	,,	*—	*—	±	—	—	—
		159–159·5	,,	*±	*±	—	—	—	—
		158–158·5	,,	*+	*—	±	±	—	—
		156–156·5	1·5	+	+	NT	NT	NT	NT
		154–154·5	>3<4	+	+	NT	NT	NT	NT
G	Naturally infected milk+ ‘Henzerling’ yolk sac suspension	163–163·5	<1·5	—	—	NT	NT	NT	NT
		162–162·5	,,	—	—	NT	NT	NT	NT
		161–161·5	,,	—	—	NT	NT	NT	NT
		160–160·5	,,	—	—	NT	NT	NT	NT
		159–159·5	,,	—	—	NT	NT	NT	NT
		158–158·5	,,	—	—	NT	NT	NT	NT
		156–156·5	,,	—	—	NT	NT	NT	NT
		154–154·5	>2·5<3	—	—	—	—	—	—

Key to Table I

+ =serologically positive guinea-pig.
± =serologically doubtful positive guinea-pig.
— =serologically negative guinea-pig.
NT=not tested.
♀=unheated milk result is included here because of the phosphatase; infectivity tests on other unheated milks are contained in Table II.
* =passage of liver and spleen suspension to other guinea-pigs did not cause infection.
★=This milk gave a high value (1·8) after boiling in a repeat test; ? trace phenol in the sample.

TABLE II

R. burneti infectivity titres of unheated milks used in H.T.S.T. and laboratory pasteurization experiments

Unheated milk used in	Dilution of milk that infected guinea-pigs*									
	10^{0}	10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}	10^{-6}	10^{-7}	10^{-8}	10^{-9}
H.T.S.T.—										
Exp. A ...	+ +	+ +	— —	NT	NT	NT	NT	NT	NT	NT
B ...	+ +									
C ...	+ +	+ D	+ —	NT	NT	NT	NT	NT	NT	NT
D ...	+ +									
E ...	+ +	NT	+ +	+ D	+ +	+ +	— —	— —	— —	— —
F ...	+ +	NT	+ +	+ +	+ +	+ +	+ —	+ —	— —	— —
G ...	+ +	NT	NT	+ ±	— —	— —	— —	— —	— —	— —
Laboratory pasteurization—										
Exp. A ...	+ +	+ +	+ +	+ +	+ —	— —	NT	NT	NT	NT
B ...	+ +	+ +	+ +	+ +	— —	NT	NT	NT	NT	NT

+ = serologically positive guinea-pig.
+ — = serologically doubtful positive guinea-pig.
— = serologically negative guinea-pig.
D = guinea-pig died.
NT = not tested.
* The dilutions of milk were inoculated into the guinea-pigs in a dosage of 3 ml.

TABLE III

Effect of temperatures on infectivity in the laboratory pasteurization test

Experiment	Milk exposed for 30 minutes to			Guinea-pig inoculation results			
	Mean Temperature °C.	Extreme range °C.	Mean Temperature °F.	Guinea-pig number 1	2	3	4
A	65·0	64·9—65·1	148·8	—	—	NT	NT
	64·5	64·0—64·8	148·1	—	—	NT	NT
	63·3	63·0—63·5	145·9	—	—	NT	NT
	63·1	62·8—63·3	145·5	—	—	NT	NT
	62·0	61·8—62·0	143·5	D	—	NT	NT
	61·5	61·2—61·6	142·7	+	—	NT	NT
	60·0	59·7—60·1	139·8	+	+	NT	NT
	56·1	56·0—56·8	132·7	+	+	NT	NT
B	62·6	62·4—62·7	144·6	—	—	—	—
	61·7	61·6—61·8	143·1	—	—	—	—
	61·1	60·8—61·2	141·9	—	—	—	

+ = serologically positive guinea-pig.
— = serologically negative guinea-pig.
D = guinea-pig died.
NT = not tested.

A rather surprising result of the H.T.S.T. experiments was the temperature at which the phosphatase activity was reduced below 1·5 Lovibond blue units. Treatment of the milk at 158°F. for 15 seconds consistently reduced the phosphatase value to less than 1·5 L.B.U., and a variable amount—less than 1·5 to 3·0 L.B.U.—remained after treatment at 156°F. Though it is well-known that milk treated in commercial H.T.S.T. plants at 161–163°F. has a negative phosphatase reaction, we have been unable to find any consistency in the experimental determinations of the temperature at which the phosphatase is converted from positive to negative in the H.T.S.T. process. Thus, Andersen *et al.* (1948), using a special technique in which phosphatase concentrate and heated phosphatase-negative milk were mixed and held at calculated intermediate temperatures, found that phosphatase was inactivated in 1·25 minutes at 154·4°F. and in 20·5 seconds at 159·0°F. Sanders and Sager (1948), using a laboratory pasteurization apparatus, found, on the other hand, that 1 minute at 157°F. and 24 seconds at 160°F. was required to inactivate the phosphatase. Hiscox (1944), using two commercial plants of an older design than that used in our experiments and a temperature of 158°F., found that the " holding time " necessary to inactivate the phosphatase varied from 15·5 to 21·5 seconds. In addition, exposures to temperatures of 156°F. and 154°F. for 15 seconds gave, in two instances, phosphatase values of the same order as those obtained in the present experiments. The relevance of the laboratory results to those obtained using modern commercial plants, is perhaps, questionable, but is the only information available.

In the present experiments, the phosphatase appears to have been more rapidly inactivated at 156°F. to 158°F. than in the experiments of the observers mentioned above. The plant used was operating below its normal rating and the possibility was considered that the milk might have attained its holding temperature on the milk plates before it emerged into the holding tube. However, consideration of the heating rate curves at a flow rate of 48–50 g.p.h. and at various hot water-milk temperature differences showed that this was not so. It would seem that at the high temperatures used in the H.T.S.T. process, small variations in the holding time or the rate and method of heating to the holding temperature may make a considerable difference to the degree of inactivation of phosphatase, and until the present results are confirmed they should be interpreted with caution.

The combination of positive guinea-pig inoculation and negative phosphatase results in experiments E and F is of interest because Huebner *et. al.* (1949) isolated *R. burneti* from two phosphatase-negative milks which had been commercially pasteurized by the vat-holding method. The present experiments suggest that this is likely to occur with the H.T.S.T. method only when the number of rickettsiae in the milk is large in proportion to the amount of phosphatase.

The minimum legal temperatures and holding times for pasteurization at present required in Great Britain are 161°F. for 15 seconds in the H.T.S.T. process and 145°F. for 30 minutes in the vat-holding method* (Statutory Instrument No. 1589, 1949). The experiments reported in this paper suggest that with naturally infected milk these temperatures are likely to be effective in making the milk non-infectious. If, however, the number of rickettsiae in the milk is large—10^5 guinea-pig infective doses per ml. or greater—the margin of safety is diminished in both methods of pasteurization, especially with the temperature (161°F.) used in the H.T.S.T. process. Although milk from a single infected cow might contain as many as 10^5 guinea-pig infective doses per ml., it is improbable that the titre of bulked herd milk would often attain this value. A few determinations of the infectivity titres of the herd milk of Farms R and W (Marmion and Stoker, 1950) showed the milk to be infective at dilutions up to 10^{-2} but not higher. More observations are required on this point, however.

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* Since this paper was submitted for publication Ransom and Huebner (1951) have described laboratory experiments showing that two strains of *R. burneti* were able to survive heating for 30 minutes at 63°C. (145·4°F). The inocula used in these experiments contained more rickettsiae than those used in the experiments described in the present paper.

The survival of *R. burneti* at various time and temperature combinations in these experiments and in those of Huebner *et al.* (1948) suggests that the organism is rather more heat-resistant than *Mycobacterium tuberculosis* and that the temperature, 161°F., at which the tubercle bacillus is consistently destroyed in the H.T.S.T. process does not offer so great a margin of safety in dealing with *R. burneti.*

Summary

Milk naturally infected with *R. burneti* and milk with added *R. burneti* has been treated at various temperatures in a small commercial high-temperature short-time pasteurization plant.

Treatment of naturally infected milk at 154–156°F. for 15 seconds was sufficient to render the milk non-infective for guinea-pigs. Treatment of the artificially infected milk containing large numbers of rickettsiae at a temperature of 158–159°F. for 15 seconds was not successful in destroying *R. burneti* which was, however, inactivated at 161°F. for 15 seconds.

Milk with added *R. burneti* was tested in a laboratory pasteurization experiment and it was found that exposure to a temperature of 143·5–144°F. for 30 minutes rendered the milk non-infective for guinea-pigs.

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1

TEN YEARS OF DIPHTHERIA IMMUNISATION

David Thomson, M.D., D.P.H., Ministry of Health

It is now ten years since the national campaign for the immunisation of children against diphtheria was launched by the Ministry of Health. The change which has taken place in the prevalence of the disease during the decennium has been most striking. In 1941, 50,797 cases of diphtheria were notified and of these 2,641 were fatal, whilst for the preceding ten years the average annual number of deaths was 3,115 and that of notifications was around 60,000. During 1950 the number of cases had fallen to 980 with 49 deaths,* and this satisfactory position has been maintained into 1951, when, in the first quarter, only 8 deaths* were notified. The comparative figures for 1950, 1949, 1948, 1947 and 1946 were 24, 29, 61, 80, and 200 respectively. The case-fatality rate has shown no commensurate decline, despite some diminution in the early years of the campaign. In 1941 it was 5·19 per cent. and in 1950 it was 5 per cent. This disappointing fact may perhaps be due to the present rarity of the disease causing delay in diagnosis and treatment.

The following figures, compiled from reports submitted to the Ministry of Health, clearly show, however, that the prognosis for those who contract the disease, but who at some time have been immunised, is much better than that of those who have never had any such protection.

Year	Number of cases in children under 15 years	Number of these patients who had had full courses of immunisation	Number of deaths under 15 from diphtheria	Number of deaths in children who had had full courses of immunisation	Case fatality rate per 1,000	
					Non-immunised	Immunised
1945...	12,514	4,431	593	36	68·9	8·1
1946...	7,719	2,723	325	15	62·0	5·5
1947...	3,941	1,287	198	16	68·6	12·4
1948...	2,431	788	126	6	73·0	7·6
1949...	1,322	435	63	4	66·5	9·2

(A few districts did not make returns.)

1949 is the last year for which detailed figures of immunisations are available. In this year the case-fatality rate for non-immunised children under 5 years, in the country as a whole, was 11·3 per cent. and for immunised 2·3 per cent. The corresponding figures for the age group 5-15 were 3·8 per cent. and 0·33 per cent. In the county boroughs non-immunised children aged 0-5 years had a case-fatality rate of 11·7 per cent. and immunised children one of 3·2 per cent., whilst the figure for non-immunised children aged 5-15 was 1·8 per cent. and for immunised 0 per cent. In the county districts the case-fatality rates were 11·7 per cent. for non-immunised children aged 0-5 years and 1·6 per cent, for immunised children ; in the age group 5-15 years the ratio was 5·7 per cent. compared to 0 per cent.

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* Provisional figure.

The attack rates per 100,000 child population under 15 for 1949 are shown in the following table.

Area	Immunised		Non-immunised	
	0–5 years	5–15	0–5 years	5–15
Whole Country	6·8	7·0	18·8	37·1
County Boroughs	10·5	9·1	31·1	55·8
Counties	5·2	6·2	13·4	29·2

A feature of the age incidence of diphtheria during the past ten years has been that adults have been relatively more often attacked. A likely explanation is that, with the general reduction in the number of clinically recognisable and inapparent infections, older persons, because they no longer, or only infrequently, have contact with the organism, are more vulnerable when they are exposed. Support has been given to this view by the higher proportion of Schick positive persons that has been found in the older age groups, although, admittedly, it is being increasingly appreciated that the Schick Test is not a very accurate method of assessing the degree of immunity.

This decrease in natural immunity amongst women has been reflected in the increase in the ratio of Schick positives in children under six months. As a result, active immunisation of mothers in the seventh or eighth month of pregnancy has been advocated, and is carried out quite extensively in the United States. Opinion in this country doubts the value of the procedure, since the antitoxin, transferred by way of the placenta, has only a transient effect and may markedly reduce the value of later active immunisation of the infants.

Notifications were not divided into age groups until 1944, but a comparison between the age distribution in that year and in 1950 is set out in the following table. These figures show that the fall in incidence continues to be less in the youngest and oldest age groups, as commented on by W. J. Martin.(1)

Age	Notifications per 1,000,000		1950 as a percentage of 1944
	1944	1950	
0—	1,378	72	5·2
5—	2,561	80	3·1
10—	1,558	52	3·3
15—	579	24	4·1
25—	88	6	6·8

It has long been appreciated that diphtheria is more prevalent in females than males over the age of 15, and this preponderance of female cases has been noted in the United States, France and Germany as well as in Britain. These ratios have, however, declined sharply in this country in the six years, during which more detailed grouping of cases has been carried out. The following table gives the number of female cases for every male.

Year	1944	1945	1946	1947	1948	1949	1950
Aged 15–24	2·56	2·31	2·55	2·21	2·23	2·41	1·92
25 and over	3·62	3·38	2·11	2·08	1·94	1·98	1·79

There has been considerable debate as to the reason for this higher proportion of female cases. Some maintained that there was a higher degree of female susceptibility. In France, where compulsory immunisation of Army personnel was instituted in 1936, this protection was put forward as an explanation, until the fact that the position was one of long standing was pointed out. In view of the relative decline in the past decade, it may be that the intensity of exposure is a significant factor. Women, as mothers or elder sisters, are more likely to come in contact, through nursing, with sick children, and, as well as a decline in diphtheria cases in children, there has been a fall in the number of carriers. Again, a contributing reason for the sex disproportion in the early years of the period was that many men were in the forces.

It is noteworthy, however, that, although the incidences of scarlet fever and measles have altered little during the seven years, a similar trend has occurred in these infections. In the case of whooping cough the decline has not been anything like so marked. Previous infection cannot be the explanation, since, in early life, both sexes are affected with these diseases roughly equally. The female preponderance in the age group 25 and over is shown in the table.

	Ratio of Females to Males, aged 25 and over						
Year	1944	1945	1946	1947	1948	1949	1950
Scarlet Fever	2·55	2·64	1·88	1·58	1·38	1·46	1·15
Whooping Cough ...	3·90	4·98	3·18	3·47	2·74	3·13	3·04
Measles	3·26	2·40	1·97	1·86	1·63	1·62	1·61

In the course of the past decade 9,148,005 children under 15 years have been immunised against diphtheria and 2,145,095 secondary or reinforcing injections have been given, under official schemes. In addition, some children have been immunised privately. By the end of the war, it was calculated that just over 60 per cent. of the child population had had anti-diphtheria vaccine injections, compared to around 8-10 per cent. before the nation-wide campaign began. This ratio mounted until, in 1949, it was estimated that 66 per cent. of children were protected.

Unfortunately, in 1950, for the first time, there was a sharp decline in the number of primary immunisations carried out, as shown below.

Yearly average 1946-1950 = 633,744.
1949 = 693,441.
1950 = 517,436.

This last year's total falls much below that necessary to maintain the high percentage of children protected which had already been achieved. The greatest reduction was in the older children. In 1950, 75·4 per cent. of the 1949 total of children under five years were immunised, but only 70·7 per cent. of those aged 5-14. The number of re-inoculations also decreased in 1950, the total being 383,071 compared to 500,485 in the previous year.

The falling off in immunisations was particularly marked in the second half of the year (total primary immunisations for first half year = 305,529 ; second half year = 211,907), and this decline is even sharper than the figures suggest, since, in previous years, more immunisations have been carried out in the second six months than in the first. It seems to have been caused, to a large extent, by the fear that, during times of poliomyelitis epidemics, inoculations may tend to precipitate a paralytic form of that disease. This problem is a difficult one; a special committee has been appointed by the Medical Research Council to enquire into it, and investigations have been planned under their direction. Thus it may be some time before the degree of risk incurred in diphtheria inoculations when there is a high incidence of poliomyelitis in the immediate locality can be accurately assessed. For the present, all that can be said is that a prima facie case has been made out for the possibility during the epidemic prevalence of poliomyelitis of some occasional connection between recent immunisation and the onset of paralysis. This applies alike to diphtheria and combined diphtheria—whooping cough immunisation, and to injections for other purposes (e.g. penicillin), which, along with other traumata, such as tonsillectomy, are under suspicion, of sometimes precipitating an attack of paralytic poliomyelitis.

On the other hand, present evidence strongly suggests that the risk of immunisation when poliomyelitis is not rife in a neighbourhood is infinitesimal. Analysis of the immunisation returns from the various local health authorities for the second half of 1950 is striking. The decline in the numbers immunised was not only marked in districts, where poliomyelitis was epidemic, but also in others where the incidence was little more than what has now to be accepted as normal. Again, it was noticeable that in some places, where the respective incidences were similar, quite different policies appeared to have been adopted towards immunisation.

During 1950, when over half a million immunisations were carried out, only 58 cases of poliomyelitis, developing within four weeks of injection, were reported to the Ministry, and in only 46 was the recently injected limb affected. With such a number of children immunised in a year in which approximately 5,000 cases of paralytic poliomyelitis were notified (70 per cent. of which were in children under 15) there is obviously room for coincidence. But, admitting that the majority of these 58 cases (and perhaps some others which were not reported) were probably not merely coincidental, no opinion can be expressed on the significance of these figures, save that the risk appears to be a very small one. Every effort should be made to ensure that the highly successful immunisation campaign is not brought into disrepute, by an over-emphasis on this risk, so small in comparison with that of a recrudescence of epidemic diphtheria.

Again, it would be doubly unfortunate if the very success of the campaign led to a decreased demand for immunisation. This risk was clearly shown in Miss G. Rowntree's report on the inquiry carried out in March, 1948, concerning diphtheria immunisation in a national sample of children aged two years.(2) It was revealed that 80·2 per cent. of first children, and only 50·9 per cent. of fourth or higher order, children had been immunised. Admittedly, such a factor as the increasing preoccupation of the mother with a large family may help to explain this position, but it would seem that a certain indifference towards the risk, after the older protected children had escaped infection, was a contributory factor. It cannot but be potentially dangerous that this should be the position in that part of the child population which has most contacts and is generally most overcrowded.

It has long been appreciated that, unless the proportion of immunised population is kept up to the 70 per cent. level, protection against epidemic diphtheria cannot be reasonably assured. This conclusion was strongly supported by the Medical Research Council Report(3) which dealt with diphtheria outbreaks of an unusually severe form, which occurred on Tyneside and Dundee during the war years.

As the authors point out, the danger now is that the proportion of protected persons may drop to a level at which not only the population of a few unfortunate towns might fall victims to the disease, but also large parts of the country would be vulnerable. For, whereas vaccination against smallpox becomes effective in a few days, immunity against diphtheria takes several weeks to develop. The only members of a community exposed to diphtheria who can expect protection—at short notice—apart from those receiving the temporary, and less dependable, protection from therapeutic serum, are those who have been inoculated earlier in life. With the decline in incidence of diphtheria, and the consequent decrease in the number of those who develop immunity through subclinical infection, the significance of inoculation in providing the means of protection is greatly increased.

Admittedly, the morbidity and mortality rates for diphtheria have been falling steadily since the beginning of the century—in 1901 the death rate per million children under 15 was 888, whilst in 1938 it was 301 and in 1949 under seven. This decline suggests the operation of other factors besides artificially induced immunity, such as better hygiene and improved methods of treatment. The type of diphtheria bacillus present may be decisive. It has been suggested that there is a cycle of evolution of C. diphtheriae.(4) This might be mitis-intermedius-rare intermedius-gravis-gravoid-mitis. The various types may run true over periods of years and mutations take place under various unknown stimuli.

However, in reviewing the first decade of the national immunisation campaign, during which the incidence of diphtheria has fallen so dramatically, it is salutary to recall that, once at least before in this country, the disease was comparatively rare—and then got out of hand. Sir John Simon, when he first assumed the post of medical officer to the Privy Council, in 1858, wrote—"diphtheria is a disease which (though it has been experienced in former times) is well-nigh unknown to the existing generation of British medical practitioner". Care must be taken that no transient difficulty or minute risk in the carrying out of the immunisation campaign allows history to repeat itself.

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MUMPS AND DIABETES

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I. *Mumps and diabetes mellitus*

Pancreatitis is an occasional complication of mumps, and changes in the pancreatic Islets of Langerhans are found in association with diabetes mellitus. Not unnaturally, therefore, the suggestion has been made from time to time that mumps may play a part in the causation of diabetes (e.g. Medical Officer, 1949). Gundersen (1927) noted an increase in deaths from diabetes at ages 10-20 in years following epidemics of mumps. He mentioned that as early as 1864 Stang had reported a case of diabetes occurring during convalescence from mumps, and that Harris had described another case in 1899. Cole (1934) studied 18 cases of diabetes among children and found that mumps had been a prelude in three instances. He gave references to several other cases reported in the literature.

The cases of diabetes reported as following mumps have mostly been of an acute type occurring in childhood, and the onset of diabetes has followed closely upon the attack of mumps. The possibility must be considered, however, of other cases of diabetes developing so slowly and insidiously after mumps as to conceal any connection between the two diseases. Such a connection would be strongly suspected, if it were found that a history of mumps was given by persons suffering from diabetes appreciably more often than by the non-diabetic population.

To test this possibility a special question about mumps was introduced into the regular monthly Survey of Sickness carried out by the Social Survey on behalf of the Registrar General (for further details about the Survey of Sickness see Stocks, 1949, or Logan, 1950). Between August, 1949, and July, 1950, 43,041 adults aged 16 and over, interviewed in connection with the Survey of Sickness, were asked at the end of the routine interview "Have you ever had mumps?" The answers were classified as "definitely yes", "probably yes", "don't know", "probably no", and "definitely no", and in the first two categories the age or ages at which mumps was said to have occurred was recorded. If the subject asked what sort of disease mumps was, a simple description was read to him as follows :—

> "Mumps is a catching disease. It is usually young children who get it but occasionally grown-up people do. There is a swelling of the glands on one or both sides of the face just below the ear and above the angle of the jaw. The swelling lasts a few days, and it may hurt to try to open the mouth wide. There is no rash."

The purpose of the special question was not explained to the subject by the interviewer.

The results were tabulated according to whether or not the subject reported that he had been suffering from diabetes during the previous two months and are shown in Table 1. As the number suffering from diabetes was small (152 out of 43,041 adults), a broad age grouping was adopted, viz. ages 16-64 and 65 and over. An effect of this broad age grouping is to introduce some negative correlation between mumps and diabetes : older people are more likely to have diabetes than younger but, as will be seen below (Table 6) are more likely to forget having had mumps. A finer age grouping might have helped to solve this problem but would have introduced other difficulties.

TABLE 1

Distribution of replies to the question " Have you ever had mumps ?"

		Definitely Yes	Probably Yes	Probably No	Definitely No	Don't know	Total
Males, 16–64 ...	Diabetics	8	2	6	13	9	38
	Others ...	4,179	1,170	3,034	5,680	2,794	16,857
Males 65 and over.	Diabetics	1	1	3	5	2	12
	Others ...	295	157	540	841	628	2,461
Females 16–64	Diabetics	19	5	8	18	5	55
	Others ...	7,093	1,346	2,673	6,925	2,023	20,060
Females 65 and over.	Diabetics	12	2	6	17	10	47
	Others ...	751	253	621	1,188	698	3,511
All persons 16 and over.	Diabetics	40	10	23	53	26	152
	Others ...	12,318	2,926	6,868	14,634	6,143	42,889

The results in Table 1 can be looked at from two points of view. Table 2 presents diabetes rates among persons giving or not giving a history of mumps. Men showed a negative correlation between diabetes and mumps, that is to say, diabetes was rather less frequent among those who gave a history of mumps. The differences are not statistically significant and, as has been said, are explainable by failure of older men to remember having had mumps. Elderly women, however, seem to have a better memory for mumps, and among women the correlation between diabetes and mumps was positive, though not significantly so.

TABLE 2

Diabetes rates per thousand persons interviewed.

	Said they had had mumps. (Definitely or probably)	Said they had *not* had mumps. (Definitely or probably)
Males, 16–64	1·87	2·18
65 and over	4·41	5·76
Females, 16–64	2·84	2·70
65 and over	13·75	12·55
Persons, 16 and over	3·27	3·52

TABLE 3

Percentage of persons interviewed who said they had definitely had mumps.

	Persons with diabetes	Persons without diabetes
Males, 16–64	21	25
65 and over	8	12
Females, 16–64	35	35
65 and over	26	21
Persons, 16 and over	26	29

Table 3 gives the results the other way round, showing the percentage of diabetics and of other persons who claimed a definite history of mumps. None of the differences between those with and those without mumps was significant. As would be expected, the correlation was again negative among men, while among women it was positive only in the older group.

The conclusion to be drawn from the results in Tables 1-3 is that there is no evidence of a real relationship between diabetes among adults and a history of mumps. Adult diabetics seem to be no more likely to have had mumps some time in the past than are persons not suffering from diabetes.

II. *Incidence of Mumps by sex and age*

As mumps is not a notifiable disease, information about its incidence is far from complete. Therefore some of the other results of this special mumps survey, although based on more or less remote memories of childhood, are worth placing on record. No differentiation will now be made in regard to diabetes.

TABLE 4

Proportions who said they had had or had never had mumps.
(Percentage of all persons interviewed, by sex and age).

	Definitely Yes	Probably Yes	Probably No	Definitely No	Don't know	Total per cent.	Number
Males, 16–64 ...	25	7	18	34	16	100	16,895
65 and over	12	6	22	34	26	100	2,473
Females, 16–64 ...	35	7	13	35	10	100	20,115
65 and over	21	7	18	34	20	100	3,558
Persons, 16 and over	29	7	16	34	14	100	43,041

Table 4 gives the distribution of all persons interviewed according to their replies about having had mumps. Twenty-five per cent. of men and 35 per cent. of women under 65 were quite sure that they had had mumps at some time, and a further 7 per cent. of each sex thought they had probably had it. At ages 65 and over 12 per cent. of men were sure and 6 per cent. thought it likely that they had had mumps, the corresponding proportions of elderly women being 21 and 7 per cent. Just over a third of all those interviewed were quite definite that they had never had mumps, and this proportion was practically the same in each sex-age group.

Table 5 analyses in detail the sex-age distribution of the attacks of mumps reported by those whose answer had been classified " definitely yes ", and from these figures attack rates have been calculated and are given in Table 6.

TABLE 5

Age-distribution of attacks of mumps of every person whose reply was " definitely yes " (including multiple attacks).

Age of Attack

Age at Interview	0–	5–	10–	15–	25–	35–	45–	55–	65–	75+	Not stated	Total
Males												
16–	184	474	153	47							80	938
25–	152	517	160	61	24						122	1,036
35–	111	449	189	64	43	33					145	1,034
45–	71	317	129	39	25	18	15				116	730
55–	43	161	79	21	18	12	2	7			50	393
65–	30	88	41	9	8	6	4	2	—		45	233
75+	9	26	24	7	3	—	1	—	—	—	10	80
Not stated	3	4	4	2	1	—	—	—	—	—	4	18
Total	603	2,036	779	250	122	69	22	9	—	—	572	4,462
Females												
16–	222	728	225	91							102	1,368
25–	192	951	404	123	73						214	1,957
35–	151	738	336	119	93	54					235	1,726
45–	102	583	264	101	55	53	24				226	1,408
55–	65	336	178	65	42	32	11	15			166	910
65–	20	209	86	40	30	14	8	3	4		115	529
75+	10	72	49	14	6	11	1	2	4	1	70	240
Not stated	1	4	—	—	1	1	—	—	—	—	1	8
Total	763	3,621	1,542	553	300	165	44	20	8	1	1,129	8,146

TABLE 6

Attack rates of mumps per thousand persons interviewed (persons answering "definitely yes" only).

Age of Attack

Age at interview ...	0–	5–	10–	15–	25–	35–	45–	55–	65+
Male									
16–	80	205	64	*39*					
25–	43	146	45	17	*13*				
35–	30	122	51	17	11	*17*			
45–	24	108	44	13	7	6	*10*		
55–	19	72	36	9	8	5	1	*6*	
65–	21	63	30	6	6	4	3	1	—
75+	13	40	36	9	4	—	1	—	—
All ages	35	120	46	14	7	4	1	1	—
Female									
16–	76	248	77	*59*					
25–	44	218	90	27	*31*				
35–	37	181	80	26	22	*25*			
45–	30	170	74	28	15	14	*11*		
55–	24	125	66	22	13	11	4	*10*	
65–	11	116	46	20	16	7	4	2	*4*
75+	11	82	55	14	7	11	1	2	4
All Ages	37	176	74	25	14	8	2	1	0

Notes:

1. Figures in italics relate to groups whose experience was incomplete; the rate has been adjusted to compensate for this.
2. Cases without statement of age have been rateably distributed over the age-groups.
3. Only the first of multiple attacks are included.

In general, the incidence reported by elderly persons interviewed was much lower than that reported by younger persons. As no secular trend of incidence of mumps could reasonably account for these age differences, there can be little doubt that they arose mainly from the natural failure of many elderly people to remember about the illnesses they had suffered from in childhood. The differences were greatest in regard to mumps under the age of five. When mumps was said to have occurred at 5-9 elderly women seemed to remember about it much better than elderly men. In each sex the reported attack rate was highest at ages 5-9, next at 10-14, and third at ages under 5. At ages over 5 the rates for females were usually considerably higher than for males.

Twenty attacks of mumps were reported in infancy (5 male, 15 female, Table 7), and 3 of these were in the first month of life. At the other extreme the oldest case reported was in a woman of 78.

TABLE 7

Attacks reported at ages under 1 *year* ("*definitely yes*" *only*).

Months

	0–	1–	2–	3–	4–	5–	6–	7–	8–	9–	10–	11–	Total under 1 year
Males	1	—	—	1	1	—	1	—	—	1	—	—	5
Females	2	2	1	2	2	—	3	1	—	1	—	1	15

TABLE 8

Multiple Attacks (" definitely yes " only).

	2 attacks	3 attacks	More than 3 attacks	Total persons	Total attacks
Males	31	2	—	33	68
Females	104	17	2*	123	278

* 4 times and 15 times.

One hundred and fifty-six persons (33 males and 123 females) claimed to have suffered from mumps more than once (Table 8). Two women each claimed more than three attacks : one was quite sure that she had had mumps in four successive years at ages 10 to 13, and the other asserted that she had had mumps every year from 5 to 19. The 154 others made more credible claims—135 said they had two attacks and 19 said they had three.

The material in Table 6 provides a means for estimating crudely the proportion of people likely to have suffered from mumps at a given age, and these estimates are in Table 9. On the assumption that the lower rates normally reported by persons interviewed in the higher age groups were due to failure of memory, two series of percentages are shown, the first, designated " high ", based on the highest age-specific attack rates reported for any age (usually those given by persons aged 16-24 or of the current age-group) and the second series, designated " low ", based on the average age-specific rates of all persons interviewed. These estimates, it should be noted, ignore the replies classified as " probably yes ", of which some would certainly relate to genuine attacks of mumps. These, however, may be offset by cases erroneously included in the " definitely yes " category. The true incidence probably lies somewhere between the " high " and " low " estimates, and it would be unreasonable to attempt a narrower estimate on the evidence available. On the " high " estimate* 35 per cent.

TABLE 9

Estimated per cent. of persons claiming to have definitely had mumps by the age stated.

	Age in years							
	5	10	15	25	35	45	55	65
Males—								
" High "...	8	29	35	39	40	42	43	43
" Low " ...	4	16	20	22	22	23	23	23
Females—								
" High "...	8	32	40	46	49	52	53	54
" Low " ...	4	21	29	31	33	33	34	34

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* The Report on " Epidemics in Schools " Med. Research Council, Spec. Report 227, p. 127, gives the percentage of boys, who have had mumps before entry (usually at 13½ years) at 33 per cent. and similarly that for girls at 20 per cent.

These figures relate to some 8,000 well-to-do boys and 5,000 girls, observed over 5 years in boarding schools. Bransby (1951) in a report not yet published on " Absence from school " with a population of more than 9,000 children found that 45 per cent. had had mumps before the age of 12 years (personally communicated). (*Ed.*)

of males would have had mumps by age 15, increasing to 43 per cent. by age 65; the more conservative estimate gives 20 per cent. by age 15 and 23 per cent. by 65. Proportions are much higher among females—40 per cent. by 15 rising to 54 per cent. by 65 as "high" estimates; and 29 per cent. by 15 and 34 per cent. by 65 as "low" estimates. Stating the results even more broadly, perhaps about a quarter of boys and a third of girls will have had mumps by the age of 15 and altogether about a third of men and a half of women will have suffered from mumps at some time during their lives.

Summary

I. To test the hypothesis that mumps may be a cause of diabetes mellitus, the question "Have you ever had mumps?" was inserted in the regular Survey of Sickness the field work of which is carried out by the Social Survey on behalf of the Registrar General.

Analysis by the General Register Office of 43,041 replies revealed no evidence of association between diabetes among adults and a history of mumps.

II. The replies classified as "definitely yes" were used to calculate attack rates of mumps by sex and age. It is suggested that perhaps a quarter of boys and a third of girls will have had mumps by the age of 15 and that about a third of men and half of women will have had mumps at some time during their lives.

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NOTIFICATION OF INFECTIOUS DISEASES IN ENGLAND AND WALES, MAY, 1951

Issued from the General Register Office, Somerset House, W.C.2.

	May 5th	May 12th	May 19th	May 26th	Average weekly figures for May 1950
Scarlet Fever	1,032	1,027	992	933	1,266
Whooping Cough	3,483	3,206	2,561	3,002	3,227
Diphtheria	45	38	31	43	49
Measles, excluding Rubella ...	18,310	19,933	18,440	19,189	7,957
Acute Pneumonia	588	574	483	613	530
Meningococcal Infection ...	27	33	36	41	37
Acute Poliomyelitis (Paralytic)...	17	11	14	22	21
Acute Poliomyelitis (Non-paralytic)	4	10	10	8	10
Ophthalmia Neonatorum ...	33	32	29	28	41
Puerperal Pyrexia and Puerperal Sepsis	81	78	75	99	80
Dysentery	833	940	661	689	408
Paratyphoid	7	10	10	15	9
Typhoid	4	3	6	5	5
Smallpox	0	0	0	0	0

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

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A STUDY OF DIPHTHERIA IN TWO AREAS OF GREAT BRITAIN

A summary of Medical Research Council Special Report No. 272

P. Hartley, Kt., C.B.E., M.C., D.Sc., F.R.S., W. J. Tulloch, O.B.E., M.D., M. Anderson, M.D., W. A. Davidson, M.D., J. Grant, M.D., W. M. Jamieson, M.D., C. Neubauer, M.D., R. Norton, M.B., and G. H. Robertson, M.D.

Introduction

It has been suggested to us that workers primarily concerned with the practice of immunization against diphtheria would welcome a summary of the main conclusions reached in our study of diphtheria on Tyneside and at Dundee. In the following pages an attempt has been made to meet this request. Our original Report must, of course, be consulted for the information on which our conclusions are based.

In both areas clinicians, bacteriologists and serologists had worked independently and in ignorance of the results being obtained by one another, either within their own or the other area. They came together only at a late stage of the work and had almost completed their observations before it was appreciated how similar their objects had been, and how much the final results, obtained by different methods and approach, had in common or were complementary, so that, taken together, they were mutually supporting and much more impressive, interesting and convincing than either alone.

One reason for selecting Tyneside and Dundee for these studies was that in each area, despite an intensive and well-conducted campaign which had led to the active immunization of over 50 per cent. of the juvenile population, highly virulent diphtheria was prevalent and at times more than one-third of the admissions on Tyneside, and one-half of those at Dundee, occurred in persons who had received a full and apparently satisfactory course of inoculations of prophylactic.

The work was begun to discover, if possible, why some of these inoculated persons developed clinical diphtheria. Although in the inoculated diphtheria was nearly always mild, and was never fatal, the fact that the disease occurred at all in such persons appeared anomalous and suggested that the scheme for the active immunization of the people, then being advocated as a national policy, contained defects which inquiry might reveal and subsequent research remedy.

The antitoxin level in inoculated diphtheria patients

Controlled experiments on Tyneside, in which special care was taken in collecting the samples of serum and the possibility of contamination had been eliminated, proved beyond doubt that the titre of circulating antitoxin in a certain number of immunized persons who contracted the disease was at the time of their admission to hospital largely in excess of that usually considered adequate for protection against the disease.

On Tyneside 25 of 95 (26 per cent.) of this type of patient had 0·1 unit per ml. (u/ml.) or more of antitoxin in the early stage of illness ; at Dundee this was true of 29 of 199 (15 per cent.). On the other hand only 7 (5 per cent.) of the 141 non-inoculated diphtheria patients on Tyneside had 0·1 u/ml. or more of antitoxin on admission ; 100 (71 per cent.) had no detectable antitoxin, in contrast to only 18 of 95 (19 per cent.) of the inoculated persons with the disease.

The question arose as to whether the titre of antitoxin in these patients was high at the time of the onset of the disease or whether it was low at that time (which would account for the patients contracting the infection and would be more in accord with the usual ideas and teaching) and subsequently increased. Examination of serial samples of serum from inoculated persons who contracted the disease, and who had not been Schick-tested or given antitoxin as a therapeutic measure, showed that the titre was in fact low at the onset, increased rapidly over several days sometimes to phenomenally high levels, and slowly declined over a period of months. The results from seven inoculated patients and two patients who had suffered from a previous attack of the disease are summarized in Figure 1. They show that the antitoxin level is low at first but that the immunity mechanism responds well and fairly rapidly under the stimulus of infection and, together with the results of a similar investigation of more seriously ill patients, they provide a reasonable explanation for

(*a*) the relapse to the Schick-positive state of persons who had suffered an attack of diphtheria from which they had recovered ;

(*b*) the occurrence of second attacks of the disease ;

(*c*) the mild nature of the illness as it occurs in inoculated persons ;

(*d*) the frequent recovery of inoculated patients without the administration of therapeutic antitoxin.

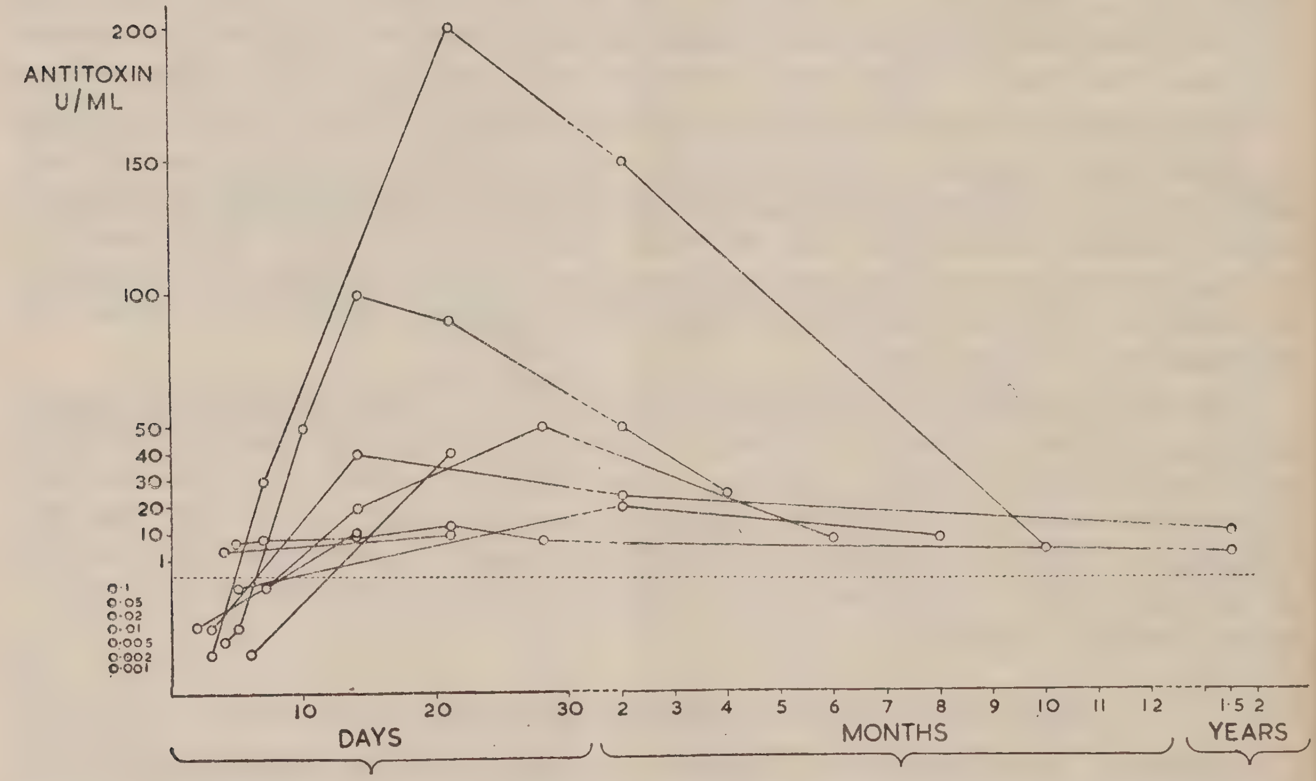

FIG. 1.—Changes in the antitoxin concentration immediately following admission to hospital, during early and late convalesence, and subsequently, of seven inoculated patients and two patients suffering a second clinical attack, none of whom received antitoxin therapeutically.

Though the authors of the Report are satisfied with the validity of this explanation of the apparent paradox that gave rise to the inquiry, namely the frequent occurrence of clinical diphtheria in fully inoculated persons, attention must be drawn to the following anomalies, which suggest a doubt whether immunity and susceptibility to diphtheria are explicable solely in terms of antitoxin.

On Tyneside it was observed that:—

(*a*) in uninoculated patients the early therapeutic administration of further heterologous antitoxic serum in large amounts sometimes failed to bring early relief and to avert death ;

(*b*) nurses with little or no circulating antitoxin did not, on taking up duty in diphtheria wards, necessarily fall victims to the disease ;

(*c*) antitoxin was often absent, or present only in very low concentration, in the serum of familial contacts who nevertheless remained well.

From both areas instances were reported of persons who had not been inoculated, whose serum contained little or no antitoxin, and who remained well, although harbouring fully virulent *gravis* strains.

At Dundee anomalies of another kind were noted: cases were reported of:—

(*a*) patients having the same, or similar, inoculation history, and with the same titre of circulating antitoxin who differed greatly in the severity of the attack from which they suffered ;

(*b*) patients who responded by the production of widely different amounts of antitoxin exhibiting degrees of severity of illness in no way correlated with the antitoxin titre of their serum. Thus, in nine cases, although there was little or no circulating antitoxin, the attacks were of the " mild faucial " type, while there were three with high antitoxin titre who suffered from " severe nasopharyngeal " infection.

The effect of prophylactic inoculation on the course of the disease

It must be specially emphasized that the case fatality among inoculated patients was *nil* whereas among the uninoculated it was 11·0 per cent. in 1943 and 5·9 per cent. in 1944 on Tyneside, and 9·5 per cent. between October 1941 and October 1942 in Dundee.

The advantage enjoyed by the fully inoculated patients was not confined to survival ; their illness was very much milder. Among the non-inoculated severe pharyngeal and nasopharyngeal cases of the malignant or hypertoxic type were common, with early development of nasal obstruction and " bull-neck ", along with a guttural " gagging " of the voice ; facial pallor and oral foetor soon became evident, and in the earliest stages of the illness swelling of the fauces was accompanied by a thin gelatinous exudate of the tonsillar surface that spread rapidly as a film over the swollen area and progressively thickened to form a typical membrane which stripped with difficulty and left behind a ragged bleeding surface. Death resulted from circulatory or cardiac failure, sometimes accompanied by haemorrhages. In the severe cases that survived the acute stage, cardiac and nervous complications, often severe, were common.

Among the cases of diphtheria that arose in fully inoculated persons the symptoms were very different. The nasopharyngeal cases were fewer, as the exudate was usually restricted to the tonsillar surface. In many patients the exudate was far from typical and was often distributed in follicular spots or patches. Cervical adenitis was much less pronounced, and paralytic and cardiac sequelae were rare and not so severe.

On Tyneside a feature of the cases occurring in the non-inoculated was the progression of the local changes and the frequency of death even when therapeutic antitoxin had been administered early in the illness. The inoculated patients apparently responded promptly to antitoxin, but, since writing the main Report, the authors have wondered whether this prompt response was not due more to the production of homologous antitoxin in the inoculated patient, consequent upon the stimulation of his immunizing mechanism as the result of infection, than upon the antitoxin administered therapeutically.

The effect of therapeutic antitoxin

In both the Tyneside and Dundee epidemics the absence of effect, or the delay and slight effect, of very large doses—50,000 to 100,000 or more units—of therapeutic antitoxin given early in the disease was a disturbing feature which suggested either infection with an unusual strain of the organism, the pathological effects of which were not provided for in the antitoxin, or some defect in the antitoxin itself ; both of these factors may have been operative. In both epidemics almost all the cases were accounted for by a particular strain of *gravis* organism, closely similar if not identical in the two areas ; and it is interesting that, independently, each group of workers suggested the possibility of its possessing enhanced virulence or unusually invasive properties. In reviewing the position it must be recalled also that practically all the antigens used for prophylaxis and antibodies used for treatment throughout the world are derived from one strain of *C. diphtheriae,* namely P.W. No. 8. Said to have been originally a *mitis* strain—" atypical " would perhaps be a more apt description now—it was isolated by Park in 1895 from a mild or moderate case of diphtheria. It has been subcultured many thousands of times but has always been, and remains, a notable toxin producer.

Further, the method used to immunize animals for the production of antitoxin and the preparation of that agent for therapeutic use have undergone many changes during the past half-century. To begin with, animals were immunized by injecting toxic filtrates ; these have been replaced first by filtrates whose toxicity has been modified, and later by highly purified preparations of such filtrates. The first antitoxin used clinically for therapy consisted of the whole serum separated from the blood of immunized animals. Later the crude serum was processed to remove much of the inactive protein. During the last ten years highly purified antitoxins have been prepared by peptic digestion of the natural serum. These modern preparations are of very high potency and produce so little reaction on injection that a large number of units can be given in small volume by any route ; they neutralize toxin, they flocculate, and, when they are injected, antitoxin is found in the blood of the recipient. It has frequently been pointed out, however, that with increasing purity of the product it has been necessary to give larger and larger doses to produce the same clinical effect. It has been reported from some areas that administration of large doses of purified antitoxin has not been followed by the expected clinical improvement and it has, therefore, been suggested that by modern methods of preparation and purification antitoxin may be deprived of useful, and even essential, components. If the original crude antiserum should be lacking in essential components it may be that processing emphasizes this lack and therein may be the cause of the failure, or delay, in clinical effect leading to the need for injection of increasingly large doses. One of the authors of the Report has shown that crude diphtheria antitoxin which passes the placenta of pregnant guinea-pigs readily and in abundance, and in small doses sensitizes anaphylactically the whole animal or the isolated uterine plain muscle, loses both these properties on being subjected to peptic digestion (Hartley, 1948).

Practical Conclusions

Though the authors of this Report approached their studies in a critical spirit, they remain in no doubt as to the importance of active immunization, and they *insist* on the necessity of this in early life. In spite of there being solid grounds for satisfaction in the results achieved, there are none for complacency and the effort must be maintained. This is specially so at the present time when there are very few cases of diphtheria and, therefore, the means whereby man acquires a natural active immunity—by sub-clinical infection—is temporarily in abeyance. If effective prophylactic is injected the child is not only assured of a concentration of circulating antitoxin which greatly reduces the possibility of the disease being acquired and practically abolishes the chance of infection terminating fatally, but is provided with an immunizing mechanism which, as this work has clearly shown, is no less important than the antitoxin that is the early product of its action. This latent mechanism responds either to a subsequent injection of antigen or to exposure to infection, so that a person who has been inoculated as a child can expect to secure *for himself* early and adequate protection should he be exposed to infection in later life. If in later life a reinforcing dose of antigen is given, there is a notable rise in antitoxin titre, as is shown by the recent results of Balch (1950) published at the same time as our Report. Even if a reinforcing dose of antigen is not given and the patient is exposed to infection, the results of these studies show that the infection itself serves as a secondary stimulus, so that antitoxin is rapidly produced, very high titres are attained, and the disease is mild with almost invariable recovery.

It cannot be adequately emphasized that it is the bounden duty of parents to ensure that their children are actively immunized, *whether diphtheria is or is not prevalent*, and preferably before the end of the first year of life. Persons so treated can confidently expect protection in early life and also possess the means of acquiring it without delay in later life. It must never be forgotten that active immunity against diphtheria cannot be acquired in a few days but takes some weeks to develop, so that those who have been denied prophylactic inoculation in good time have no such assurance of safety.

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ACCIDENTS AMONG YOUNG CHILDREN

Abstract of a paper on "Accidents among Children under Two Years of Age" by G. Rowntree, M.A., published in the Journal of Hygiene (48, *III, Sept.* 1950) *and summarised here by kind permission of the Editor of that Journal.*

The literature on the subject of child accidents and injuries suggests that young children are exposed to particularly serious hazards, especially burns. It is therefore important to discover what is the national incidence of child accidents, and what are their chief causes. The paper, of which this article is an abstract, discusses these questions in the light of some findings of a national inquiry carried out with the aid of a grant from the Nuffield Foundation by a Joint Committee of the Royal College of Obstetricians and Gynaecologists, the Population Investigation Committee and the Institute of Child Health (University of London). The results of this inquiry were based on questionnaire data collected in 1946 and again in 1948 by the health visitors of 422 local authorities in Great Britain with a view to studying many aspects of the health and development of a national sample of about 15,000 children who had all been born in one week, 3rd-9th March, 1946. The methods used in this inquiry and the response obtained from the families (91 per cent. of the women concerned co-operated), have been described in detail in earlier publications and will not be dealt with further here.*

Data available on accidents

At the 1948 interview each mother was asked whether her two-year-old child had ever had either a burn or scald, a bad cut or bruise, or broken a bone. If any of those accidents had occurred she was asked to say how many months old the child was when it had happened, what part of the body had been injured and who had treated the injury. The health visitors were instructed to inquire how the accidents had occurred, and to enter the causes briefly on the questionnaires.

In designing the 1948 inquiry it was realized that the memory factor might affect the reliability of the survey information, especially as the data on accidents occurring during a two-year period was collected only at the end of that period. Though mothers would be likely to remember serious accidents involving hospital and medical treatment, their ability to recall minor injuries, not requiring professional care, probably varied greatly. Complete accuracy on every detail cannot be claimed in an extensive national investigation, but the survey data appear to be sufficiently reliable for an estimate of the national incidence of accidents to be made.

The incidence of accidents

The incidence of accidents among children under two years could only be calculated on the basis of the total number of children surviving to the second birthday, and therefore each exposed to the risk of accidents for the full period of 24 months. The survey data on the accidents occurring to children who died before they were two years old was in any case incomplete since 30 per cent. of the mothers of these dead children were not interviewed.

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* c.f. *Monthly Bulletin of the Ministry of Health*, vol. 9, June 1950, pp. 134–136; also *Maternity in Great Britain*, Oxford University Press, 1948, pp. 1–14.

A total of 348 surviving children of two years of age had had either a burn, a scald, or bad cut or bruise or a fracture which had been treated by a doctor or in a hospital department. This total represents an accident rate of 79·6 per 1,000 children. Another 262 children (57·5 per 1,000) were reported to have had slight injuries for which medical treatment was not obtained. As mentioned above, the possibility of forgetting slight accidents seemed considerable, and for this reason the incidence of minor injuries given here is probably a less reliable estimate than the figures given for treated accidents. Consequently, the following discussion is devoted chiefly to an analysis of the latter.

The types of injuries sustained by survey children are shown in Table I according to the source of treatment. The eight children who have been seriously injured twice, sustaining, for example, both a burn and a cut, are counted twice in this table, once in the burns column and again in the column for cuts and bruises. The total incidence of injuries which can be calculated for this table is therefore slightly higher than the rates given above for children injured.

TABLE I

Incidence of Accidents by Source of Treatment

Source of treatment	Injuries per 1,000 children		
	Burns and scalds	Bad cuts and bruises	Fractures
Hospital:			
Accident or other ward	5·1	0·2	1·2
Out-patients' department	7·6	13·0	4·9
General practitioner	24·4	23·5	1·4
All sources of treatment	37·1	36·7	7·5
Not treated	35·5	26·3	—

General practitioners provided treatment for burns and for cuts very much more often than hospitals. As for hospital treatment, cases of bad burns and scalds were usually admitted to accident wards, serious cuts and bruises being treated chiefly in out-patients' departments. All the fractures recorded received professional care, chiefly at out-patients' departments.

The rates given in the table can be used for calculating very approximately the total annual incidence of these serious accidents among young children. It must, of course, be remembered that the table refers only to the group of children reaching their second birthdays in March, 1948, and not to all children under two years of age at that date. This difficulty can to some extent be overcome by the application of a life-table technique to the survey data. When this is done, the survey data show that during one year doctors and hospital departments treated about 15,000 severe burns or scalds, 16,000 bad cuts or bruises, and 4,000 fractures among an estimated total of 1,740,000 children under two years of age in Britain in 1947. This was before the National Health Service Act came into force. Over 35,000 injuries to very young children must have been treated annually, 3,000 in accident wards, 11,000 in out-patients' departments and 21,000 by general practitioners, involving an enormous amount of work for hospital and medical services.

Causes of accidents.

Most of the injuries to children under two years occurred in the home. Among the treated injuries there were only three resulting from motor accidents, and four others which took place away from the children's usual surroundings, i.e. the house or the garden. The survey data were analysed in order to discover whether there was a close association between poor housing conditions and high accident rates among children under two years of age, but it was found that a bad housing environment (as measured by such items as overcrowding and lack of domestic amenities) was not an important factor, since it was not associated with a particularly high accident rate.

The fact that children under two years of age were equally exposed to risks of accidents whatever their home conditions may possibly be explained by the stages of development through which they were passing in the early years of life. While still immobile and sleeping for most of the day, the risks of injury were slight, being largely limited to the possibility of rolling off the bed or out of the cot or being burnt, while lying on the hearth rug, by a live coal spurting from the fire. But when the children were beginning to walk, they were exposed to many more risks, whatever kind of home they were living in. They might trip against or fall off furniture, and tumble down steps or into the hearth. Growing more curious as well as more mobile, they would begin to prod and touch strange objects, and might put their fingers in hot food, cut themselves on knives or broken glass or touch live coals or naked flames.

The chief causes of the serious medically treated accidents in the present sample are shown in Table 2, together with the age or stage of development at which they occurred. The considerable variety of causes and the small numbers involved have made some grouping necessary in this table. Scalds from boiling kettles and hot cups of tea were combined, and so were burns caused by various items of domestic equipment, cooking stoves, coppers, boilers, cooking utensils, hot food and irons. Electric and gas heaters as well as open coal fires were included under the heading " domestic fires ". The burns from other causes included those due to inadequately protected hot-water bottles, lighted cigarettes, and playing with boxes of matches. Various causes of cuts and fractures were also combined: falls were grouped under two headings, first those from cots, high chairs and beds which could mainly be attributed to lack of supervision at the time of the accident, and secondly those which occurred when the child was moving about, i.e. falls against and off furniture, and down steps. Cuts caused by sharp instruments and by broken glass were combined with cases where children's fingers were trapped, either in doors or in household equipment, such as the mangle. Other causes of cuts and fractures included falls from heights (one child fell out of a window, another fell down a 30 ft. pit in a runaway pram), car accidents (in one case a child had crawled under a stationary vehicle), and bites by dogs and by a tame rabbit.

Table 2 shows the number of accidents due to each cause in relation to the total number of months during which each child was exposed to the risk of accident at each stage of development. The total number of accidents in this table is small. Nevertheless, the incidence by cause and age shows a slight significant increase when the children started sitting-up, and a much more marked and significant increase in each type of injury after the children began to walk about. The only exception was in the case of falls from prams or cots where the increase was not significant. As children became steadier on their feet the rates for most accidents showed a decline. Burns from domestic fires were an exception to this rule, probably because of seasonal variations, in the use of fires

TABLE 2

Frequency of injuries receiving medical treatment by cause and age of occurrence

Age of child when accident occurred	Treated injuries per 10,000 months of exposure										All treated injuries
	Burns or scalds					Fractures, cuts or bruises					
	Due to spilling hot drinks	Due to domestic fires	Due to other domestic equipment	Due to other causes	All causes of burns or scalds	Due to falls from pram or cot	Due to falls elsewhere	Due to knives, other sharp edges and to trapped fingers	Due to other causes	All causes of fractures, cuts and bruises	
Before sitting-up ...	0·3	1·0	0·7	1·3	3·3	1·0	—	—	0·3	1·3	4·6
Sitting, but not yet walking	2·7	3·0	1·8	1·5	9·0	2·7	3·6	0·9	1·5	8·7	17·7
Already walking:											
Up to 16 months ...	18·8	6·5	9·8	—	35·1	6·5	22·1	7·3	0·8	36·7	71·8
17–20 months ...	6·8	2·2	6·8	1·7	17·5	2·8	23·1	8·5	2·8	37·2	54·7
21–24 months ...	4·3	10·3	3·8	1·1	19·5	0·5	12·4	7·6	2·2	22·7	42·2
All ages	4·7	3·9	3·5	1·3	13·4	2·3	9·2	3·7	1·4	16·6	30·0
Total number of accidents	53	44	39	14	150	26	103	41	16	186	336*
Percentage of all accidents due to different causes ...	15·8	13·1	11·6	4·1	44·6	7·7	30·7	12·2	4·8	55·4	100·0

* Twenty cases of injury have been omitted from this table, eighteen because the cause was unknown, and two because age or stage of development at the time of the accident was not fully recorded.

for space heating. Most of the survey children were beginning to walk in the spring and summer months of 1947 (they were 16 months old in July of that year) when fires were seldom lighted. The period for which the incidence of burns from this cause was highest, i.e. when the children were 21-24 months old, coincided with the coldest winter months, November, 1947–March, 1948, when domestic fires would be burning all day.

Few mothers were able to isolate their active children from the chief domestic hazards by leaving them under separate supervision in rooms away from the household routine. Only 10 per cent. of the mothers interviewed had full-time help with the housework, while a further 9 per cent. received part-time or occasional assistance. Over four-fifths of the survey mothers had no domestic help at all at the date of interview, and were therefore obliged to do the various household duties, cleaning, cooking, washing and shopping, with the active infant following at their heels during his waking hours. In these circumstances some accidents were inevitable, and many could in fact be attributed entirely to these circumstances. For instance, one mother accidentally trod on her baby's finger when he was playing on the stairs and another woman was making up the fire when her child fell on the hot poker.

Although infants and young children were exposed to many unavoidable risks while learning to walk and playing in the home, some of the hazards could have been removed. Few mothers could be accused of deliberate neglect but many women may have been unaware of all the dangers which existed for toddlers in their own homes. The clause in the Children's Act (1908) which penalizes parents whose young children have been badly burnt through the failure to protect open fires is largely ineffective, except in so far as it has helped to publicize the necessity for adequate fireguards. Health visitors are vigilant in this matter, and in describing causes of burns on the questionnaires some of them reported that guards were not used. But it is difficult to enforce the ruling on fire guards, when for instance, women have to use old-type kitchen ranges for cooking as well as space heating.

Even the most modern houses are not always designed to give the maximum protection to young children. Lack of space, insufficient shelving, and poor design of kitchens may result in infant injuries, while the increasing provision of domestic aids and labour-saving devices, particularly electrical equipment, mean even greater hazards for the inquisitive child. More adequate protection is required and more instruction should be given to mothers on the dangers which exist in every home.

Summary

Among a national sample of young children studied in 1946-8, the rate for all serious injuries (i.e. those receiving professional treatment) during the first two years of life was 79·6 per 1,000. Serious cuts were almost as frequent as burns, but there were few fractures. General practitioners were responsible for treating the great majority of these injuries.

Almost all accidents occurred in the home, but the accident rate in these early years of life was not significantly affected by the quality of housing conditions. Most accidents happened when the children were still unsteady on their feet or had not yet developed " heat-sense ". Few mothers had domestic help and accidents often occurred because the children had to play in the kitchen while mothers were doing their cooking and housework.

THE FOOD AND NUTRITIONAL SITUATION IN BERLIN DURING THE BLOCKADE AND AFTER. PART I

W. T. C. Berry, M.B., P. J. Cowin, M.R.C.S., D.P.H., and H. E. Magee, M.B., D.Sc., M.R.C.P., Ministry of Health

The blockade of Berlin was established towards the end of June, 1948, and at the request of the Foreign Office on 20th September two of us went there to advise as to what immediate nutritional measures were required for maintaining the health of the Berliners in the Western Sectors, and, having done this, to plan to keep systematic observations at fairly regular intervals on a representative sample of the population. This paper is based on our study of the ration scales in the circumstances of the blockade and on the observations we made on the sample. The persons in the sample were examined by two of us according to a pre-arranged plan at the time of our second visit in November, 1948. Essentially the same procedure was followed on subsequent visits; January, March, and October, 1949. All three of us were present at the last two visits, but only two in January.

On the last four occasions we were assisted by Dr. Borgmann, a Fellow of the Koch Institute.

Sources of Food. At the time of our first visit Berlin had been practically a beleaguered city for about ten weeks, and the official rations were, therefore, to be regarded as the only important source of food. There were two minor sources (*a*) gardens and allotments, and (*b*) smuggling from the Russian Sector and Zone. The quantity under (*a*) was estimated at about 20,000 tons, which, reckoned as potato, almost certainly an over-valuation, would provide only about 2½ ozs., or 40 calories, per head daily for four months for the population of the Western Sectors, and as they probably did not last as long, were obviously too small to take into account. There was also no alternative but to ignore the insignificant amount smuggled over the border, of which the distribution was very uneven. Our enquiry, however, made it clear that the amount of food so gained made no appreciable difference to the 2·25 millions in the Western Sectors as a whole.

The Air Lift

The first question was whether the Western Air Forces were taking full advantage of dried foods to secure maximum economies in bulk and weight. It was quite clear that this was being done. We then considered whether more energy could be lifted by any change in the character of the foods, e.g., increase in fat, while keeping the air lift constant. We reckoned that by such a change the addition which could be brought in would be no more than 70 calories per head daily, even by increasing the fat ration to the furthest procurable limits. Increase in the food lift therefore became inevitable if improvements in the diet were to be effected.

Pattern of Diet. The dietary pattern exhibited by the original rations was a strange one. (Table 1.) It was in many ways contrary to physiological principles. For instance, the rations for the child of 0–1 years provided 1,786 calories, of 1–6 years 1,653, of 7–9 years 1,619, and of 10–14 years 1,559 calories. The trend should, of course, have been in the opposite direction to a level of some 3,000 calories or so for boys aged 14 and over. Expectant and nursing mothers were fairly well provided for, but there was no differentiation in favour of males during adolescence. The types of persons classed as "heavy workers". Group I (2,500 calories) included many who were not working full time and many, e.g., clerks, administrators, actors and trade union officials, who were sedentary or light workers; in

TABLE I

Food rations for Berlin in grammes daily until 1st November, 1948 : Changes then made in brackets

	Bread	Potatoes	Cereals	Meat	Fat	Sugar	Cheese	Milk	Calories	
									Pre Nov., 1948	Adopted Nov., 1948
Heavy Workers, Group I* ...	600	400	80	100	30 (40)	25 (40)	— (5)	—	2,498	(2,609)
Workers—Group II	500	400	60	65	15 (30)	20 (40)	— (5)	—	1,999	(2,202)
Employees—Group III ...	400	400	40 (50)	40	10 (30)	20 (40)	— (5)	—	1,608	(1,882)
Children up to 6—Group IVa	300	400	30	20	20	25	— —	Up to 1 yr. ·75 litre	1,786	(1,786)
								1–6 yrs. ·5 litre	1,653	(1,653)
Children 7–9—Group IVb ...	300	400	35	20	23 (25)	40	— —	·25 litre	1,619	(1,633)
Children 9–14—Group IVc ...	300 (350)	400	40	20 (40)	25 (30)	50	— (5)	—	1,559	(1,834)

* Group I limited to 4 per cent. of population.

Mothers placed in Group II from 5th month till end of pregnancy and given 500 c.c. milk from 4 months before till 4 months after labour.

addition many of these could obtain an extra meal of 900 calories on five or six days a week. Group III (1,608 calories) on the other hand included many persons who should have been in Group II (2,000 calories) or even in Group I.

Qualitatively the diet was no less strange, and perhaps the strangest thing was the absence of milk from the rations of everyone over the age of nine years, except expectant and nursing mothers. The main sources of energy in the diet were bread and potatoes and they were also the bulkiest elements. Potatoes were in dried form, which limited the ways in which they could be cooked and served. The amounts of meat, fat and sugar were small ; the term " meat " included the rough energy equivalent in the form of fish, usually canned, or dried egg. There was no cheese or fresh vegetables. For a European accustomed to a varied mixed diet, one more drab and uninteresting would be difficult to imagine. The usual school meal or the extra meal for the heavy workers was almost always a thickish porridge made of macaroni, semolina or similar cereal preparations (Nährmittel), and sometimes sweetened with sugar.

The intake of food for each person from ration sources averaged about 1,800 calories per day, which is, of course, so low that to live upon it necessitated modifications in behaviour and in way of living. Over the previous three years the citizens of Berlin had had experience in the art of restricting activity according to intake, and the effect could be seen in the behaviour of people in the street. They walked with a slow, deliberate gait. Purposeless movements were avoided. There was an absence of spontaneity, of bustle and life, very noticeable in contrast with people in the cities of Western Germany. Children, in particular, lacked the vivacity of well-fed children.

People wore very many clothes, even on warm days. Some appeared so immersed in their own thoughts that they would walk across the road looking neither to right nor left, so that violent brakings were almost daily experiences for all who drove cars.

Increased Rations Recommended

In September, 1948, increases in the rations amounting to about 200 calories for Groups I (heavy workers) and II (workers) about 300 for Group III (other employees) and 400 for Group IV*c* (children 9–14 years) were recommended. All males 14–20 years were to be placed in Group I and all females of the same age in Group II ; the school meal of 250 calories was to be given as an extra to *all* school children without exception ; all over nine years were to get a ration of five grammes cheese daily and of dried skimmed milk. Regard was, of course, paid to the availability of the foods and the capacity for increase in the air lift. The adoption of these recommendations would have involved an increase of 120 tons a day in the air lift ; the extent to which they were adopted called for an increase of 98·5 tons. This is shown for individual items in the two sets of figures in Table 1. A ration of dried milk was not adopted for anyone over nine years, or the special arrangements for adolescents.

In view of the small amounts, or complete lack, of foods rich in calcium, e.g., milk, cheese and green vegetables in the diet, our recommentions included (*a*) the addition of calcium carbonate at the rate of 14 ozs. per 280 lbs. to flour intended for bread-making, and (*b*) the issue of cod liver oil to every child below the age of six. Both these recommendations were put into operation in March, 1949.

The ordinary consumer's rations provided about 0·17 gm. of calcium daily, and from analyses of the Berlin water supply we were able to make an estimate of the amount of calcium consumed in water. We placed the average daily consumption in the region of 0·24 gms. of calcium daily, thus making a total of 0·41 gms. daily for the ordinary consumer. This is only 0·04 gms. less than the amount generally regarded as just sufficient for the adult. The requirements for calcium as for other nutrients are known to vary widely and the human organism has considerable powers of adaptation, so that, on the whole, it seems unlikely that the Berlin population suffered from calcium shortage.

The vitamin C content of the diet was expected to be very low because all the vitamin C-bearing foods were dried or processed. Samples of the rations were analysed by the Government Chemist in London. Contrary to expectations it was found that the dried potatoes and dried cabbage included in the rations contained appreciable amounts of vitamin C. We also had to take into account that, for so long as they lasted, fresh vegetables from the farms on the outskirts of Berlin were issued to the public at the rate of 2 kg. per month. The calorie value of these vegetables, like that of dried vegetables, was deducted from the issue of potatoes, so that they were not an addition to the energy value of the diet. As we did not have figures for the amounts of vegetables included in the ration and as the vitamin C content of the different brands of dried potato varied widely, only a very rough calculation of the vitamin C consumption was possible. Probably it was seldom less than 5 mg. or more than 10 mg. per head daily from natural foods. To this must be added a variable but appreciable consumption of ascorbic acid tablets by persons whose numbers, through the vigorous efforts of the Public Health Department, had by March increased so far as to comprise some 80 per cent. of the population of the British Sector. We had been informed in September that a weekly ration of 150 mg. of ascorbic acid was to be provided. We therefore made no recommendations about vitamin C. Whatever the precise consumption was it can hardly have been seriously low because we did not see any clinical signs of scurvy anywhere in Berlin.

The increases recommended in September, 1948, were meant to tide the population over a relatively short period and were not considered adequate for a long period of stress. Because of the continuance of the blockade at the time of our fourth visit in March, 1949, we pressed for the adoption in full of our original recommendations, slightly modified, to make all the diets conform better with requirements. The major recommendations were:—a ration of milk for everyone, extra rations (Zuschläge) to male adolescents and expectant mothers, of 213 and 580 calories respectively, increases of from 100 calories for adults to 300 for children 6-9 years old and a reduction of 450 calories for children under one year. None of these recommendations was put into operation, but they were agreed to, and would no doubt have been applied if the blockade had not ended on May 12th.

Fuel, Light and Clothing. The amount of solid fuel available for household heating and cooking was meagre, only a small fraction of the household ration in Great Britain during the time of our greatest shortage of coal in the winter of 1946-47. In every house, apartment, or institution visited during the blockade there were complaints of insufficient coal and wood for heating and cooking. Those who cooked with coal gas were no better off, for enough pressure for burning was to be had only seldom and at irregular times. In no household which we visited was solid fuel used for heating only ; it was always burnt in the kitchen for cooking, and as far as possible, when all the family were at home so that all could benefit from the heat. Towards the end of the blockade community space heating in large halls was instituted. The arrangement was gaining in popularity when the weather began to warm and the blockade was removed.

Lack of artificial light was only less severely felt than lack of fuel for heating, but the effects were measured in inconvenience, depression and annoyance rather than in physical suffering. One would have to be more than human to endure with equanimity the nuisance of electric light becoming available for no more than 3-4 hours throughout the 24 and at irregular times during the day and night. The shortages of food, fuel, soap and clothing were tangible things with which most Berliners were familiar and which they knew how to combat in some measure, but there was nothing they could do about electric light, which often came on when they did not want it and was not to be had when wanted most. This tantalising annoyance continued until the end of the blockade.

The shortage of artificial light was not without some advantage—it kept people indoors and often in bed, when otherwise they would have been up and about or outside spending precious calories to little purpose. The winter of 1948-49 was mild, and there can be no doubt that the mild weather and the enforced idleness through lack of artificial light made for appreciable economies in food energy, with the result that the nutritional state, including the weight, of the people, remained more satisfactory than it would have been if conditions had been otherwise.

With officers of the Political Division we carried out an investigation into the adequacy of clothing and footwear. Such an enquiry could only be made on a limited scale, but it was enough to give a rough idea of the position. Most people seemed to have sufficient clothing and bedding to withstand the winter (though necessity had produced some examples of great ingenuity in improvisation), but there was a proportion of " hard cases ", who were in difficulties. Some we found had to borrow clothes or footwear to leave home. As might be expected, the greatest shortage was in children's shoes ; but the proportion of cramped feet or worn-out shoes in our sample of children was small.

Sample of Population surveyed. The sample was taken from the Tiergarten and Siemensstadt Bezirke and was representative of the population from 8 years upwards. Our examinations enabled us to follow some of the effects of the changes of food supply, temperature and occupation on the physique and well-being of the population. In the sample representatives of those groups that we had learnt from our first visit were the most vulnerable—school children, adolescents, and workers in heavy industry—were balanced by similar groups of persons of the same age and sex engaged in sedentary occupations.

The total of persons in the sample in November, 1948, was 611, but no more than 600 ever turned up on subsequent visits. At the examinations of January, March, and October, 1949, additional persons of the same age and sex and of similar occupation and social status to the absentees were also examined. The observations made on these people were not included in the final appraisal of the effects on the sample in October, 1949, but they provided valuable confirmatory evidence. Of the original 611 persons selected 366 were examined on all occasions, a leakage of 40 per cent. The records of those who attended in March and those who did not, were compared for the period November-January. There was no significant difference in the weight change of the two groups. We concluded, therefore, that our findings were not affected by selection.

Medical Examinations and Measurements. The data collected included heights, weights and other body measurements, blood analyses, X-ray findings, efficiency and therapeutic tests, and clinical findings. The procedure was substantially the same at each medical examination, except that a few fresh tests were introduced in January, 1949, or later, and some others, which were considered not to be worth while, were abandoned. The advice we gave was based on our own survey findings and on the observations we made of people in the streets, at work and in other public places, from the results of our inspections of homes and institutions, and from study of the existing ration scales.

Results

Weights and Heights. The mean weights of adults showed consistent increases from November to March and from March to October for both sexes. The increases were of the order of 1-1·5 kg. for men and of 0·5-0·75 kg. for women for November-March, and 3-4 kg. for both sexes for March–October.

In Table II there are compared the mean weights and heights of the different groups of Berlin children in November, 1948, and those of London children of the same ages. All Berlin groups were lighter by some 5-13 per cent., and all but one group were shorter, than the corresponding group of London children. By October, 1949, as the same Table shows, this difference had been very much reduced, to 2-8 per cent. approximately.

TABLE II

Average Weights and Heights of Berlin Children in November, 1948, *and October,* 1949, *and of English Children* (*L.C.C.* 1948) *of the same age**

November, 1948 District	Average age Years	Weights in kg.		Difference, London minus Berlin	Heights in cm.		Difference, London minus Berlin
		Berlin	London		Berlin	London	
Tiergarten	8·4	24·1	26·5	2·4	124·1	127·0	3·0
Boys	13·4	38·3	44·0	5·7	148·5	150·0	1·5
Siemensstadt	8·0	24·2	25·5	1·3	124·3	125·0	0·7
Boys	13·9	41·0	44·8	3·8	153·5	154·0	0·5
Tiergarten	8·3	24·4	26·0	1·6	124·2	126·0	2·8
Girls	13·0	37·5	42·5	5·0	147·5	150·0	2·5
Siemensstadt	8·1	24·0	25·0	1·0	125·3	125·0	—0·3
Girls	14·1	45·1	47·5	2·4	154·5	155·0	0·5
October, 1949							
Tiergarten	9·3	26·9	28·5	1·6	131·0	131·0	0·0
Boys	14·3	44·1	46·5	2·4	155·8	157·0	1·2
Siemensstadt	8·9	27·4	28·0	0·6	130·5	130·0	—0·5
Boys	14·8	48·4	50·0	1·6	160·5	161·0	0·5
Tiergarten	9·2	26·5	28·5	2·0	130·1	130·5	0·4
Girls	13·9	43·0	46·5	3·5	152·4	154·0	1·6
Siemensstadt	9·0	26·8	28·0	1·2	130·8	130·0	—0·4
Girls	15·0	50·8	51·7	0·9	159·1	159·5	—

* From data supplied to us through the kindness of Sir Alan Daley, County Medical Officer of Health for London.

Nutritional State. The nutritional state was assessed by the procedure in use in Britain (Adcock et al (1947)), particular attention being paid to the following:—pallor, tired expression, lethargy, poor posture, diminished muscular tone and development, lack of lustre of the hair and diminished fat. By assessing these criteria and the all-round general appearance, the subjects were classified as of "good", "fair" or "poor" nutritional state. In November, 1948, many of our sample of subjects showed all or most of the above stigmata; there was slight improvement in January and in March, 1949, but in October, 1949, they had almost entirely disappeared except in a few cases.

TABLE III

Nutritional status of individuals seen in Berlin in November, 1948, *March and October*, 1949, *expressed as percentages*

Number			Good	Fair	Poor
Men ...	105	November, 1948	61·0	29·5	9·5
		March, 1949	75·2	21·0	3·8
		October, 1949	84·8	14·3	1·0
Women ...	52	November, 1948	80·8	17·3	2·0
		March, 1949	86·6	13·5	—
		October, 1949	92·3	5·8	2·0
Boys ...	98	November, 1948	42·9	46·0	11·2
		March, 1949	82·7	16·3	1·0
		October, 1949	89·8	8·2	2·0
Girls ...	111	November, 1948	64·9	27·0	8·1
		March, 1949	79·3	19·8	0·9
		October, 1949	91·9	7·2	0·9

Table III gives the proportions of the same subjects in the three nutrition grades in November, 1948, and in March and October, 1949. The amount of improvement in all groups is striking for the two periods, but especially so for the boys. Only 43 per cent. of boys attained the "good" grade in November, 1948, but 82 per cent. did so in March and 90 per cent. in October, 1949.

The least improvement was shown by the men, and more often than not men of large stature showed up badly. There seemed to be two reasons for this: (*a*) the rations were too small for the bigger men, and (*b*) an appreciable number of men were still suffering from the effects of privation in Russian camps or from the effects of war service. The women were relatively much better nourished at all three examinations. The proportions for children in October, 1949, are only a little less satisfactory than those we found for English school children during the same year; of 3,181 English children examined, 93·8 per cent were of "good", 5·5 per cent. of "fair" and 0·7 per cent. of "poor" nutritional state. (Adcock et al, 1950.)

The improvement in nutritional state, observed in October, 1949, was also seen in the general appearance and behaviour of people in the street. Indifference to traffic and deliberation of movement were replaced by alertness.

Part II will appear in the August Bulletin. It will describe the tests used and the incidence of certain physical signs of malnutrition together with a summary and references.

SURVEY OF SICKNESS—DECEMBER QUARTER, 1950

(Issued from the General Register Office, Somerset House, W.C.2)

The Registrar General's Quarterly Return No. 409 (in Tables A to H) gives detailed results of the Social Survey's interviews of random samples of the adult population carried out during the months November, 1950, to February, 1951, in which the experience of October to December, 1950, is recorded. The report given here presents some rates derived from these tables without standardization or correction for the number of days in the month, and compares them with those for preceding months. More detailed studies will appear in the publications of the General Register Office.

The figures presented in this report relating to the one month, December, 1950, are complicated by a change in the method of sampling introduced with the interviews carried out in February, 1951, i.e., the "second month" interviews concerning the December experience. Previously the samples of individuals for interview in each area had been drawn from the local records of the National Register and persons aged 16 years and over were selected. From February, 1951, the samples have been drawn from local Electoral Registers and have therefore included only persons aged 21 years and over. Thus the "second month" interviews relating to December, 1950, excluded persons aged 16-20. The resulting increase in the average age of the sample has caused a slight apparent increase, for that month only, in the various rates reported in the tables that accompany this report. This slight inflation of the December, 1950, rates is insufficient to affect seriously their comparability with those of previous months, and no correction has been made. In future reports in this series only the experience of persons aged 21 and over will be presented, rates for earlier periods being adjusted to exclude persons under that age.

The Monthly Health Index—the proportion per 100 interviewed who suffered no illness or injury so far as they remembered during the stated month—is given in Table A, separately for men and women, at ages 16-64 and ages 65 and over. The indices for October tended to be slightly lower, i.e., worse, than during October of the two previous years. Those for November and December showed no definite change.

Table B gives monthly morbidity rates (the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only), days of incapacity, and numbers of medical consultations, distinguishing persons with illness commencing in the month from those with illness continued from the previous month. Including persons with an injury, average rates for the December quarters of 1948, 1949 and 1950 compare thus:—

	Ages 16–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
Oct.–Dec., 1948 ...	68·9	103	42	84·1	273	70
Oct.–Dec., 1949 ...	68·9	98	42	82·5	267	66
Oct.–Dec., 1950 ...	69·1	96	42	84·5	179	68
Oct.–Dec., 1950 per cent. of Oct.–Dec.						
1948... ...	100	93	100	100	66	97
1949... ...	100	98	100	102	67	103

At ages 16-64 the sickness and medical consultation rates were practically the same during each of the three December quarters, but the incapacity rate diminished slightly each year. At ages 65 and over the sickness and medical consultation rates changed little, but the incapacity rate, which had been at a high level in the December quarters of 1948 and 1949, declined by about a third in the December quarter, 1950. Days of incapacity among the elderly averaged less than two days per person per month during the December quarter, 1950, in contrast with almost three days during the two previous December quarters. This was a substantial reduction and restored the incapacity rate among elderly persons to a level comparable with that prevailing in corresponding months prior to 1948. It remains to be seen whether this reduction to the former level of incapacity will prove to be permanent.

During the course of the three months October, November, and December there was some increase in rates of new illness and a reduction in continued illness. Incapacity was much higher in December than in the two preceding months.

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during the month from those who developed an illness of a mild or ill-defined nature. Average monthly rates during the December quarters of 1948, 1949, and 1950 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	16–64		65 and over		16–64		65 and over	
	M	F	M	F	M	F	M	F
Oct.–Dec., 1948	4·6	5·4	5·3	10·5	36·1	43·1	34·5	36·0
Oct.–Dec., 1949	4·4	5·9	6·5	8·6	38·6	45·1	32·7	37·3
Oct.–Dec., 1950	7·2	8·5	10·1	12·6	36·9	43·5	31·7	36·5

The proportion of persons reporting more serious illnesses was much higher in each sex-age group during the December quarter, 1950, than in either of the two previous December quarters. On the other hand, there was some reduction compared with the December quarter, 1949, in the numbers reporting minor or ill-defined conditions.

Table C shows that the increase in the more serious conditions took place chiefly in the month of December.

Table D gives average monthly numbers of new illnesses from selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the number of days of incapacity arising from each cause (including both new and continued illnesses).

Illnesses classed as colds or influenza showed practically no change in incidence during the December quarter, 1950, compared with 1948 and 1949, but there was a considerable increase in the number of days of incapacity attributed to these conditions. Other respiratory diseases gave rise to more illnesses than in the two previous December quarters, but incapacity from these causes increased only among women. Rheumatism, too, was reported more often during October-December, 1950, than in the corresponding periods of 1948 and 1949. Among elderly persons there was a large reduction in days of incapacity from rheumatism compared with October-December, 1949.

Days of incapacity from all causes (shown in the bottom right-hand corner of Table D) remained practically unchanged at ages 16-64 compared with the December quarter, 1949. At ages 65 and over where, as noted above, there was a large reduction in days of incapacity, the table shows that this reduction took place in each sex but that it was relatively much greater among elderly women than among elderly men.

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 16–64						Ages 65 and over					
	M			F			M			F		
	1948	1949	1950	1948	1949	1950	1948	1949	1950	1948	1949	1950
January	40	36	34	32	26	25	21	20	21	13	12	12
February... ...	40	34	34	31	24	27	19	18	20	15	9	11
March	42	34	38	31	24	27	23	16	20	16	12	11
April	45	39	38	34	27	29	25	19	18	15	12	11
May	45	40	41	33	29	29	27	23	21	16	15	12
June	44	43	43	34	31	31	30	24	21	19	14	12
July	45	44	41	33	32	32	26	26	22	16	14	12
August	47	45	43	34	32	32	25	26	25	15	16	11
September ...	43	45	40	31	32	29	23	25	20	16	15	13
October	39	40	36	28	27	26	24	24	22	12	14	11
November ...	36	36	38	27	27	28	22	23	22	12	13	11
December ...	35	37	34	25	28	26	17	27	20	12	14	12

(Based on combined results of interviews in two following months.)

B.—*Monthly Morbidity Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 16–64				Ages 65 and over			
	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month
1948								
January	39·8	24·6	95	38	39·2	43·6	224	64
February	39·9	24·9	105	40	38·9	43·9	239	74
March	37·4	25·8	87	41	36·4	44·4	199	67
April	34·3	26·4	73	35	35·2	45·5	152	67
May	34·7	26·0	75	35	31·7	47·8	139	61
June	34·3	27·1	71	37	32·5	44·3	108	48
July	34·9	25·9	74	36	33·8	45·3	133	67
August	34·0	25·6	69	33	32·7	48·0	163	56
September	37·6	25·2	75	35	35·4	45·6	155	48
October	41·6	25·0	101	42	40·1	42·5	193	60
November	46·0	22·3	105	43	46·2	37·0	285	74
December	47·0	23·0	103	42	44·4	41·0	334	76
1949								
January	44·5	24·5	110	46	44·2	39·7	217	72
February	46·9	24·2	131	50	44·7	41·3	207	71
March	45·9	25·2	128	46	43·7	42·1	211	83
April	39·7	27·0	84	40	38·5	46·5	175	74
May	38·2	27·1	75	40	33·1	48·1	163	61
June	34·5	28·1	77	38	32·6	47·8	142	60
July	33·4	28·4	78	41	33·1	46·6	118	63
August	32·5	28·4	74	38	32·7	46·9	71	54
September	35·9	25·2	74	39	35·9	44·6	96	60
October	44·1	22·5	93	42	40·6	40·9	212	59
November	47·1	21·0	102	41	42·8	39·4	293	73
December	51·0	20·1	100	41	46·3	36·5	323	65
1950								
January	43·9	22·5	113	42	42·2	39·7	212	68
February	46·8	22·3	117	50	43·9	40·3	211	77
March	44·7	22·2	107	49	42·2	43·3	221	87
April	41·5	24·0	86	41	42·6	43·5	183	85
May	41·1	23·5	79	42	40·5	43·9	133	80
June	37·4	25·3	78	42	36·9	47·0	94	64
July	38·0	24·9	69	37	39·5	44·1	76	65
August	38·3	23·6	64	36	37·5	44·9	104	62
September	42·4	22·7	72	40	39·8	44·1	120	73
October	47·5	21·0	83	43	44·8	39·5	137	61
November	47·3	20·0	88	42	45·5	38·4	170	70
December	50·4	19·0	117	42	48·0	35·7	227	73

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury, see Registrar General's Quarterly Returns, Table E.)

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
1948								
January ...	3·9	4·9	5·8	9·6	31·6	38·2	27·8	34·0
February ...	3·2	4·9	5·8	8·0	31·5	39·2	33·1	30·4
March ...	4·2	4·1	5·4	7·0	28·1	37·3	31·5	29·0
April	3·4	4·0	4·0	6·5	26·4	34·0	28·7	30·5
May	3·5	3·6	2·5	4·1	27·0	34·7	25·8	30·0
June	3·7	3·8	4·4	5·8	26·7	33·6	24·9	29·1
July	3·2	4·0	3·8	6·3	26·3	35·3	25·5	31·0
August ...	4·1	4·1	1·7	7·3	24·3	34·6	27·0	28·1
September ...	4·1	4·8	2·9	6·2	28·9	36·5	31·6	29·8
October ...	4·1	5·1	3·6	8·1	32·3	41·0	32·5	35·1
November ...	5·1	5·0	7·1	13·1	37·8	43·7	35·1	36·2
December ...	4·7	6·0	5·2	10·2	38·1	44·7	35·9	36·8
1949								
January ...	5·2	5·8	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	7·9	6·8	10·9	37·0	41·8	31·6	38·5
March ...	5·9	7·7	7·9	11·7	35·3	42·0	34·3	33·0
April	3·5	4·7	6·2	6·4	30·0	40·0	30·3	33·4
May	3·5	3·8	2·9	3·9	29·4	38·8	28·7	30·3
June	2·6	3·6	3·0	6·3	26·3	35·5	24·9	29·9
July	2·7	3·9	3·7	6·9	25·6	33·7	26·2	28·5
August ...	2·7	3·7	4·4	4·0	24·9	32·9	27·8	29·1
September ...	3·1	4·2	2·7	4·8	28·0	35·7	31·5	32·3
October ...	3·7	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·9	5·3	8·5	9·7	38·3	45·1	32·7	34·1
December ...	4·5	6·6	5·4	9·7	42·3	47·9	32·4	42·2
1950								
January ...	3·7	6·0	9·6	8·0	35·4	42·0	28·0	37·2
February ...	5·9	8·1	9·2	9·2	36·1	42·8	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·4	41·7	29·4	33·8
April	5·3	5·6	5·5	10·6	32·9	38·6	33·7	34·4
May	4·7	4·9	5·5	6·6	31·5	40·3	29·8	37·8
June	3·1	4·2	3·6	5·7	27·9	38·6	27·9	34·9
July	3·4	4·5	6·1	6·6	30·0	37·4	27·0	37·6
August ...	3·2	4·4	4·6	5·7	29·3	38·7	26·2	36·9
September ...	4·3	4·9	5·5	7·0	33·7	41·3	31·0	35·0
October ...	5·5	7·1	8·6	10·0	37·8	43·8	32·5	37·1
November ...	6·5	7·6	9·7	10·7	36·0	43·8	32·9	36·7
December ...	9·6	10·9	12·1	17·0	36·9	42·9	29·8	35·8

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. “ Ill-defined ” excludes all symptomatic illness which caused incapacity for work, such cases being classed to the appropriate higher category. “ Illnesses starting in each month ” include new and recurrent illnesses.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity.*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1948								
January–March... ...	14·8	16·1	11·5	11·4	12·0	15·2	12·9	14·1
April–June	9·1	8·6	5·5	7·5	4·7	6·0	1·6	8·7
July–September... ...	9·8	9·3	6·1	6·7	3·3	5·3	1·9	3·4
October–December ...	20·0	20·3	12·2	13·9	12·9	16·6	9·3	23·9
1949								
January–March... ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September... ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March... ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September... ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...	19·3	19·5	13·9	13·7	15·7	22·3	36·8	31·6
	Other respiratory disease							
1948								
January–March... ...	2·7	3·1	4·4	5·4	8·5	6·4	24·3	27·4
April–June	2·8	2·6	2·6	3·3	7·5	4·2	8·0	7·5
July–September... ...	1·8	2·7	2·7	2·6	3·3	2·4	10·1	9·6
October–December ...	3·6	3·3	4·8	4·9	9·5	7·3	24·2	22·0
1949								
January–March... ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September... ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March... ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September... ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...	5·6	6·6	7·4	7·1	15·5	15·5	35·1	48·6
	Rheumatism, all forms							
1948								
January–March... ...	4·4	6·1	7·0	8·5	9·1	7·4	19·6	12·9
April–June	4·1	5·9	8·2	7·3	3·6	2·7	4·7	16·5
July–September... ...	4·3	6·6	8·1	7·1	4·9	4·4	9·2	5·9
October–December ...	5·5	7·8	9·9	10·7	5·1	4·6	3·7	20·9
1949								
January–March... ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September... ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March... ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	8·2	22·2
July–September... ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...	6·5	9·1	9·3	13·1	4·7	5·8	6·3	16·9

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity—(continued)*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 16–64		65 and over		Ages 16–64		65 and over	
	M	F	M	F	M	F	M	F
	Disorders of Heart and Arteries							
1948								
January–March... ...	0·3	0·9	1·5	1·9	3·3	2·7	0·9	18·1
April–June	0·3	0·6	1·7	1·6	2·0	2·8	9·8	11·1
July–September... ...	0·2	0·5	1·9	2·1	1·8	2·0	17·5	16·6
October–December ...	0·4	1·0	1·2	1·2	1·2	2·2	19·5	11·5
1949								
January–March... ...	0·4	0·7	0·6	1·1	5·2	3·7	10·7	6·4
April–June	0·1	0·4	0·5	1·0	3·4	2·6	10·4	5·2
July–September... ...	0·2	0·4	1·1	1·3	1·6	3·5	12·4	7·0
October–December ...	0·2	0·8	0·6	1·5	1·7	3·8	9·5	15·5
1950								
January–March... ...	0·2	0·6	1·3	1·3	3·7	4·2	12·5	14·6
April–June	0·3	0·7	0·5	1·5	4·8	2·9	13·9	19·1
July–September... ...	0·3	0·8	1·9	1·9	4·4	2·3	10·0	7·1
October–December ...	0·4	1·2	1·5	2·2	4·0	1·4	19·5	22·0
	Skin complaints				All illnesses and injuries			
1948								
January–March... ...	1·5	1·1	2·0	0·6	110	85	203	235
April–June	1·8	1·2	0·7	0·9	85	63	128	136
July–September... ...	2·1	1·2	0·5	1·2	78	68	102	184
October–December ...	1·7	1·2	1·6	1·0	100	105	183	344
1949								
January–March... ...	1·3	1·4	0·4	0·9	119	127	200	220
April–June	1·7	1·4	0·8	0·9	84	74	151	165
July–September... ...	1·6	1·0	1·0	0·7	81	71	89	99
October–December ...	1·6	1·3	1·1	0·5	95	98	225	302
1950								
January–March... ...	1·6	1·5	1·4	1·0	111	114	196	231
April–June	1·9	1·4	0·8	0·8	88	75	143	133
July–September... ...	1·9	1·6	1·1	1·2	77	61	118	87
October–December ...	2·0	1·2	0·8	0·8	97	95	182	176

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e., new, recurrent or continued from the previous month).

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

JUNE, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

	June 2nd	June 9th	June 16th	June 23rd	June 30th	Average weekly figures for June, 1950
Scarlet Fever	1,043	972	976	857	904	957
Whooping Cough	3,220	3,277	3,291	3,440	3,533	3,363
Diphtheria	47	37	29	37	32	52
Measles, excluding Rubella ...	15,668	14,746	12,150	11,338	10,060	9,689
Acute Pneumonia	500	372	461	396	355	426
Meningococcal Infection	37	53	40	28	31	38
Acute Poliomyelitis (Paralytic) ...	16	32	25	44	41	67
,, ,, (Non paralytic)	12	20	25	35	46	20
Ophthalmia Neonatorum ...	23	40	22	39	42	40
Puerperal Pyrexia and Puerperal Sepsis	77	84	71	85	53	78
Dysentery	753	682	509	567	490	343
Paratyphoid	4	15	14	39	42	7
Typhoid	20	7	10	6	1	8
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster. S.W.1.

Northallerton Laboratory: Change of Address

The Public Health Laboratory at Northallerton has now been transferred to the Friarage Hospital (*Tel.*: Northallerton 88). Dr. D. J. H. Payne remains in charge of the laboratory.

THE STORAGE AND STABILITY OF BIOLOGICAL PRODUCTS

Brigadier A. Sachs, M.D., M.Sc.

Director of Pathology and Consulting Pathologist to the Army

1. *General*

In the Army, with its world-wide commitments, it is necessary to maintain stocks of biological products under different climatic conditions, and facilities for storage vary in efficiency in different parts of the world. The potency of biological products is dependent on the conditions of storage and the time that has elapsed since the date of manufacture. These instructions have been drawn up with the primary object of reducing wastage due to faulty storage of such products.

2. *Maintenance of stocks*

Biological products (sera, toxoids, vaccines, tuberculins, etc.) have a limited life. Stocks held by user units should therefore be kept to an absolute minimum compatible with anticipated demands in order that the item can be used before the date of expiry. Where stocks are maintained they will be stored in batches according to the date of expiry, and it is the responsibility of officers commanding distributing depots and user units to ensure that the vaccines and sera with the earliest dates of expiry are issued first. The life of biological products depends on satisfactory storage conditions as noted below.

3. *Conditions of storage*

(a) *Distributing depots*

Those in charge of distributing depots will ensure that in no circumstances is there any departure from the conditions of storage laid down in this paragraph.

(i) *Products requiring special storage*—living virus vaccines.

Vaccine lymph

Whenever possible not more than one distributing depot should intervene between the manufacturer and the user. Vaccine lymph should be obtained from the manufacturer packed in ice (at a temperature of 0° C. to minus 20° C., but no lower) and immediately stored in a refrigerator at a temperature of minus 10°-20° C. When maintained at this temperature of minus 10° C. it will keep for twelve months. This requires a special refrigerator set at minus 10° C. which is available at all medical store depots from which the lymph is distributed.

Yellow fever vaccine

Yellow fever vaccine at present supplied from the United Kingdom or South Africa must be maintained at a temperature at or below 4° C. (preferably at minus 10° C.).

(ii) *Products requiring normal storage*

Normal storage of biological products is at refrigerator temperatures of 2° C. to 10° C. The undermentioned products must be stored at this temperature:—

Toxoids. These should be stored in the dark but not at a temperature lower than 2° C. where they might freeze.

Bacterial vaccines. Particular care is necessary with alcohol-treated T.A.B. vaccine. This should never be stored at temperatures above 10° C. or below 2° C.

Rabies vaccine. The required storage temperature is as near 2° C. to 4° C. as possible, but not lower where it might freeze. Although not so labile as other vaccines, it should not be left outside the refrigerator for long periods.

All types of antisera.

Diagnostic sera and bacterial diagnostic suspensions.

(b) *User units with refrigeration*

The normal conditions of storage will be at refrigerator temperatures. All products referred to in para. (*a*) above will be stored at this temperature with the following provisos:—

(i) *Vaccine lymph* will not normally be stocked by user units ; it will be obtained fresh as and when required, and used immediately. When it is necessary to maintain stocks for short periods vaccine lymph stored at refrigerator temperatures up to a maximum of 10° C. will keep for 14 days, and must be used within this period. A suitable temperature is provided by the ordinary domestic refrigerator.

(ii) *Yellow fever vaccine* is issued only to specially selected centres for Y.F. inoculation, a list of which is maintained at the Ministry of Health.

Yellow fever vaccine must be maintained at a temperature at or below 4° C. (preferably at minus 10° C.) during storage. During transit (usually by air outside the United Kingdom) vaccine is in ice in vacuum containers. The date of expiry is three months from the date of manufacture. In view of the short life of this vaccine, it will normally be obtained by user direct from the manufacturer or distributing centre as and when required, and used immediately.

In no circumstances will yellow fever vaccine be stored other than at a temperature of 4° C. or below ; minus 10° C. is best.

(c) *User units without refrigeration*

It is possible that in certain isolated establishments refrigeration is not available. In these units only sufficient quantities of biological products will be maintained for routine use, and will be stored in a cool dark place, preferably in an ice box.

When vaccine lymph is maintained in a cool dark place (not at refrigerator temperature) it may not be used after 7 days.

Note 1. Once a capillary tube of vaccine lymph has been opened the contents must be used immediately. Any surplus must be discarded and not kept for subsequent use.

Note 2. When yellow fever vaccine has been reconstituted any solution which is not used within 30 minutes must be discarded.

Note 3. Special care must be taken in packing to ensure that the labels do not become detached from the containers of vaccines, antisera, etc.

4. *Care in transit.*

Care in the transport, collection and distribution of vaccines and sera is of the utmost importance, particularly in warm climates. Any deterioration resulting from undue exposure is irreparable. Potency, once lost, cannot be restored by subsequent cold storage. Special care is required during transit of the following:—

(*a*) *Vaccine lymph* should be packed in ice at a temperature of 0°C to minus 20°C., but not lower, during transit and on receipt immediately stored at minus 10°-20°C.

(*b*) *Yellow fever vaccine* should be packed in ice in vacuum containers during transit and on receipt immediately stored at 4°C. or below, preferably at minus 10°C.

(*c*) *Rabies vaccine* should be packed in ice and transported at temperatures as near to 2° to 4°C. as possible, but not lower where it might freeze.

(*d*) *Schick test toxin*

(i) *United Kingdom*

Provided that the toxin will arrive at its destination within 24 hours ordinary packing and transportation will suffice.

(ii) *Overseas—ex United Kingdom*

The toxin will be sent in a vacuum container packed in ice at a temperature of 0°C. The ice should be replaced at intervals of 24 to 48 hours.

(*e*) *T.A.B. Vaccine.* This vaccine, particularly the alcoholized type, should be treated in very much the same way as Schick test toxin. When sent overseas, it should be stored in a refrigerated chamber.

(*f*) *Other biological products* should be stored during transit in as cool a place as possible, and sent by the most expeditious route. Care should be taken to ensure that these products do not remain in the sun while awaiting transportation, and they should be carefully labelled to indicate their perishable nature.

5. *The life of biological products*

(*a*) The life of biological products is dependent on the conditions of storage given in para. 3 above. The life given in the table that follows is correlated with the conditions of storage. It will be noted that in this table only optimum conditions of storage are considered. Where these are not available in user units it is assumed that only sufficient quantities of biological products will be maintained for routine use, since, owing to inadequate storage facilities, deterioration and a consequent loss of potency are certain to occur.

(*b*) All biological products are kept in the dark. This directive is of special importance for materials containing thiomersalate, since preservation may become less effective if the products are exposed unnecessarily to light.

(*c*) The limits between which immunizing agents are transported and stored are reasonable and can be achieved under present-day arrangements so that medical officers may confidently assume that the life of these products, as given in the table below, is reliable.

LIFE OF BIOLOGICAL PRODUCTS

Note : Refrigerator temperature range is 2°C. to 10°C.

Name and Description of Products	Storage Temperature	Life
1. *Vaccine Lymph* ...	(*a*) Minus 10°-20°C.	(*a*) 12 months from notified date of manufacture.
	(*b*) Refrigerator temperature	(*b*) Fourteen days from date of issue.
	(*c*) Cool dark place	(*c*) Not more than seven days from date of issue.
2. *Yellow fever vaccine* ...	4°C. or below, preferably at minus 10°C.	(*a*) Wellcome: 3 months from notified date of manufacture. (*b*) S.Afr. Inst. Med. Res.: as notified by manufacturer.
3. *Toxins*		
(*a*) *Schick test toxin* ...	Refrigerator temperature but not at lower temperature where it might freeze.	(*a*) 6 months from notified date of manufacture.
(*b*) *Dick test toxin* ...	Refrigerator temperature but not at lower temperature where it might freeze.	(*b*) 3 months from notified date of manufacture.

Note.—In no circumstances is it permissible to increase the life of these products.

4. *Toxoids*		
(*a*) *A.P.T.** (preserved with thiomersalate)		
(i) Rubber-capped and stoppered.	Refrigerator temperature but not at lower temperature where it might freeze.	(*a*) (i) 2 years from notified date of manufacture.
(ii) Sealed glass ampoules.	Refrigerator temperature but not at lower temperature where it might freeze.	(ii) 2½ years from notified date of manufacture.
(*b*) *TAF** (preserved with phenol).	Refrigerator temperature but not at lower temperature where it might freeze.	(*b*) 2½ years from notified date of manufacture.
(*c*) *Tetanus toxoid** (preserved with phenol).	Refrigerator temperature but not at lower temperature where it might freeze.	(*c*) 3 years from the notified date of manufacture (given on the carton) or 6 months beyond the notified expiry date (on label of bottle).
(*d*) (i) *Staphylococcus toxoid* (1 in 10).	Refrigerator temperature but not at lower temperature where it might freeze.	(*d*) (i) 3 months from the date of manufacture.
(ii) *Staphylococcus toxoid** (undiluted)	Refrigerator temperature but not at lower temperature where it might freeze.	(ii) 1 year from the date of manufacture.

* *Note.*—Where refrigerator temperature is impracticable, store in a cool dark place. The life of A.P.T., T.A.F., tetanus toxoid and staphylococcus toxoid (undiluted) is then six months.

5. *Vaccines*		
(*a*) *T.A.B.* (Alcoholized) (or *T.A.B.C.* Alcoholized).	Refrigerator temperature but not at lower temperature where it might freeze.	(*a*) 12 months from notified date of manufacture. (This applies to vaccine obtained from Army sources).
(*b*) *T.A.B.* (Phenolized) (or *T.A.B.C.* Phenolized).	Refrigerator temperature but not at lower temperature where it might freeze.	(*b*) 12 months from notified date of manufacture. (This applies to vaccine obtained from Army sources).
(*c*) *Other bacterial vaccines* (Cholera, Plague, etc.).	Refrigerator temperature but not at lower temperature where it might freeze.	(*c*) 18 months from notified date of manufacture.

LIFE OF BIOLOGICAL PRODUCTS—*cont.*

Name and Description of Products	Storage Temperature	Life
6. *Typhus vaccine* ...	Refrigerator temperature but not at lower temperature where it might freeze.	18 months from notified date of manufacture.
7. *Influenza vaccine* (Virus vaccines).	Refrigerator temperature but not at lower temperature where it might freeze.	2 years from date of manufacture.
8. *Rabies vaccine*		
(*a*) *Lister Institute vaccine.*	Refrigerator temperature as near 2°C. to 4°C. as possible, but not at lower temperature where it might freeze.	(*a*) 6 months from date of manufacture.
(*b*) *Other preparations*...	Refrigerator temperature as near 2°C. to 4°C. as possible, but not at lower temperature where they might freeze.	(*b*) 3 months unless notified otherwise by the manufacturer.
9. *Tuberculin* (*Old*)		
(*a*) Undiluted	Refrigerator temperature ...	(*a*) 5 years.
(*b*) Diluted	Refrigerator temperature ...	(*b*) 3 months from the date of manufacture.
10. *Antitoxic sera*		
(*a*) *Enzyme-refined antitoxic sera*		
(i) Sealed glass ampoules.	Refrigerator temperature ...	(*a*) (i) 5 years from date of manufacture.
(ii) Rubber closures	Refrigerator temperature ...	(ii) 2½ years from date of manufacture.
(*b*) *Concentrated* ...	Refrigerator temperature ...	(*b*) 2½ years from date of manufacture.
(*c*) *Unconcentrated* ...	Refrigerator temperature ...	(*c*) 2½ years from date of manufacture.

Note.—For deterioration at other temperatures see Section 6 on page 176.

Name and Description of Products	Storage Temperature	Life
11. *Other biological products*		
(*a*) (i) *Concentrated bacterial suspensions for agglutination tests except Proteus OXK.*	Refrigerator temperature but not at temperatures where they might freeze.	(*a*) (i) 1 year.
(ii) *Proteus OXK* ...	Refrigerator temperature but not at temperatures where it might freeze.	(ii) 6 to 9 months from date of *receipt* in medical store.
(*b*) *Diluted suspensions*	Refrigerator temperature but not at temperatures where they might freeze.	(*b*) 2 years from date of *receipt* in medical store.
(*c*) *Diagnostic sera* ...	(i) Refrigerator temperature but not at temperatures where they might freeze.	(*c*) (i) 2 years from date of *receipt* in medical store.
	(ii) Ice-box or cool place ...	(ii) 12 months from date of *receipt* in medical store.
(*d*) *Wassermann and Kahn antigens, etc.* (products containing cholesterol).	Cool dark place NOT refrigerator.	(*d*) As notified on the labels.

Note.—The times given for the life of these reagents are intended mainly as a guide for storekeepers. Bacteriologists are advised to test the reagents from time to time by appropriate methods to make sure that they are still fully active.

Name and Description of Products	Storage Temperature	Life
12. *Blood transfusion products.*		
(*a*) *Preserved whole-blood.*	4° to 6°C. MUST NOT BE FROZEN. Do not place by the ice container in domestic refrigerator.	(i) Trisodium citrate anti-coagulant: maximum of 7 days from date of collection. (ii) Disodium citrate glucose anti-coagulant: 21 days from date of collection (blood older than this may be used in emergencies, but only on the advice of a pathologist). In both (i) and (ii) there must be a clear cut line of demarcation between cells and supernatant plasma, and no visible evidence of deterioration.

Note.—Constancy of storage temperature of preserved whole blood is of the greatest importance. Bottles may be removed from the refrigerator for a maximum period of 30 minutes for the performance of compatibility tests. Partly used bottles of blood, or bottles which have been removed from the refrigerator for more than 30 minutes, should be discarded. When transported, a temperature of 4° to 6°C. should be maintained by using a suitable insulated container.

(*b*) *Fluid plasma* ...	Room temperature (not above 22°C.) in the dark. Should not be refrigerated.	May be used provided it is crystal clear and there is no deposit.
(*c*) *Dried plasma* ...	Room temperature (not above 22°C.) in the dark.	Indefinite, provided adequately dried and sealed to exclude air and moisture (in tropical and sub-tropical climates product is canned). Only practical criterion of fitness for use is rapid (*i.e.*, within 10 min.) and complete solution on reconstitution. Incomplete solution or gel formation indicates unfitness for use.
(*d*) *Dextran*	Preferably not above room temperature.	Probably 5 years. Must be crystal clear and without deposit.
(*e*) *Crystalloid solutions*	Room temperature (not above 22°C.).	Indefinite, provided sealing is efficient. Must be crystal clear and without deposit.
(*f*) *Haemagglutinating sera.* *Anti-A and anti-B sera.*		

Control tests with known cells performed whenever these sera are used can alone show that the sera are potent and not giving false positive or negative readings. The lives given below are approximate. They may not be attained by some sera and may be exceeded by others.

(i) Dried	Room temperature (not above 22°C.).	Usually indefinite, provided adequately dried and hermetically sealed.

Name and Description of Products	Storage Temperature	Life
(ii) Liquid	Ideally at minus 22°C. ...	Several years, possibly indefinite.
	Ordinary refrigerator temperature.	12–18 months, provided sterility is maintained.
	Room temperature	Not more than 2 weeks, provided sterility is maintained. This may be increased if preservative (e.g., chloroform) has been added.

Note.—Blood-grouping serum (*liquid*) which does not contain a bacteriostatic should be kept frozen solid, preferably at a temperature below minus 22°C., and should be used with precautions against bacterial contamination and deterioration.

Anti-Rh sera:

Control tests with known cells performed whenever these sera are used can alone show that the sera are potent and not giving false positive or negative readings. The lives given below are approximate. They may not be attained by some sera and may be exceeded by others.

(i) Dried	Room temperature (not above 22°C.).	Usually indefinite, provided adequately dried and hermetically sealed.
(ii) Liquid	Ideally at minus 22°C. ...	2 years.
	Ordinary refrigerator temperature.	Up to 4 weeks, provided sterility is maintained.
	Room temperature	Few days only, provided sterility is maintained.

Notes:

(1) Sometimes the date of preparation and not the date of manufacture is given on the labels. For practical purposes these terms are to be regarded as the same.

(2) When the expiry date of the product *only* is given by the manufacturer, no extension beyond this date is permissible.

(3) When *both* the date of manufacture and the expiry date are given, the life of the product will be calculated from the date of manufacture in accordance with the instructions given in the table above.

6. *Antitoxic sera—deterioration at different temperatures*

(*a*) The rate of deterioration of enzyme-refined and concentrated sera stored at different temperatures is given in the table below:—

Type of Serum	Storage Temperature	Deterioration per annum
Enzyme-refined	0 to 5°C.	Negligible (up to 5 years).
	5 to 15°C.	Not more than 3 per cent.
	20°C.	Not more than 5 per cent.
	37°C.	10 to 20 per cent.
Concentrated	0 to 5°C.	Negligible.
	5°C.	Not more than 5 per cent.
	15°C.	10 per cent.
	20°C.	20 per cent.
	37°C.	25 to 50 per cent.

(*b*) Enzyme-refined sera (i.e., the majority of antitoxic sera including diphtheria, tetanus, staphylococcus, gas gangrene) can generally be used up to at least 5 years from the date of manufacture.

Provided that these antitoxic sera have been filled in sealed glass ampoules and stored in distributing centres at temperatures not exceeding 10°C. during the whole period of storage, the life may be extended a further two years, i.e., to seven years from the date of manufacture. Such enzyme-treated antitoxic sera received in user units from distributing centres, which have already had a life in excess of five years, will be used within six months from the date of issue and any antisera not used during this period will be destroyed.

Except in a grave emergency when more recent sera are not obtainable, these sera will not be used beyond 7 years from the date of manufacture.

When the life of a serum has to be extended, the dosage should be increased to allow for possible deterioration.

(*c*) *Sulphate-precipitated sera,* i.e., the concentrated sera, have a life of 2½ years from the date of manufacture and will not be used beyond their date of expiry except in a serious emergency, when allowance must be made for loss of unitage.

7. *Biological test products—turn-over*

(*a*) Biological test products should be turned over quickly and it should not be necessary to keep diagnostic sera in medical stores for longer than 12 months. Diagnostic sera should, however, retain their potency under refrigerator conditions in medical stores for a period up to 2 years from the date of receipt, and they should not be disposed of before that time has elapsed unless there is definite evidence that the sera have, in fact, deteriorated.

(*b*) Every effort should be made to turn over stocks of diagnostic suspensions every 6 months, and with the exception of diluted suspensions they should not be issued after more than 9 months have elapsed from the date of receipt in medical stores or other distributing units.

8. *Assay of biological products*

When reduction of potency of any biological product is suspected, representative samples should be sent for assay as follows:—

(*a*) If purchased under contract—to the Chief Inspector of Medical Supplies, Ministry of Health.

(*b*) If purchased locally—to the Chief Medical Officer, Ministry of Health.

Note: A representative sample will consist of the bottle containing the remainder of the biological product under suspicion (if available) together with two other unopened bottles bearing the same batch number. (A minimum of 10 ml. of an antiserum or 50 ml. of tetanus toxoid will be required.)

Acknowledgments

In drawing up these instructions I have been fortunate in having the advice of the Sub-Committee on Immunology of the Army Pathology Advisory Committee; Dr. H. J. Parish and other members of the staff of the Wellcome Research Laboratories, Beckenham; Dr. D. McClean and Mr. A. F. B. Standfast of the Lister Institute, Elstree; and Dr. W. d'A. Maycock, Consultant in Blood Transfusion. Their recommendations form the basis of the information and instructions given in this paper.

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THE FOOD AND NUTRITIONAL SITUATION IN BERLIN DURING THE BLOCKADE AND AFTER : PART II

W. T. C. Berry, M.B., P. J. Cowin, M.R.C.S., D.P.H., and H. E. Magee, M.B., D.Sc., M.R.C.P., Ministry of Health

Myotatic Irritability. This was sought for by pinching the subject's relaxed biceps muscle between the fore-finger and thumb by a sharp forward movement. Subjects who gave positive response showed, in the zone of stimulation, a slow contraction of the underlying muscle which appeared as a prominent ring on the front of the arm and persisted for a variable time. Where muscle tone is good, a positive response, if it occurs at all, is momentary. Responses may, however, be elicited up to 3 seconds in some subjects in well-fed populations. We recorded as " slight " responses lasting 1-3 seconds only, and as " marked " responses of 4 or more seconds. Persistence up to 8 seconds was frequently seen.

We found this myotatic irritability in about 80 per cent. of Berlin men. Its significance is not known and it has been observed in conditions as diverse as physical exhaustion and vitamin B1 deficiency. It was thought unlikely that B1 deficiency was responsible, because the bread, which contributed 50 per cent. or more of the total energy of the diet, was made from flour of high extraction, but, because of evidence brought forward by Taylor and Chuttani (1949) on this point, we carried out therapeutic trials on 8 men showing the condition. They were given daily doses of 10 mg. of aneurin for 60-150 days, but no greater improvement was observed than that in 8 controls, who also showed the condition, but were untreated.

To throw further light on the origin of the phenomenon we carried out some tests on curarised patients under anaesthesia, in St. Bartholomew's Hospital, thanks to the kindness of Sir James Paterson Ross, the Director of the Surgical Unit, which showed that the point of action of the causal stimulus is not the motor nerve or the neuro-muscular junction but the muscle fibres ; so far from disappearing, the duration of the contraction ring actually increased.

This appeared to be due to the fact that, when tested whilst conscious the subjects did not succeed in relaxing their biceps muscles to the degree which resulted under anaesthesia. It had been noted in Berlin that people who showed the most marked myotatic response also showed a remarkable degree of flaccidity of muscle, so much so that it proved possible to predict with some accuracy what the myotatic response would be, simply from the feel of the muscle. It was thought that this might represent a state of complete relaxation comparable with that found in the curarised patients. A great saving in energy would result if undernourished persons could maintain large muscle groups relaxed to this degree when not in use. The thinnest of our Berlin subjects were presumably those in whom metabolic economies were most needed, and one would therefore expect to find a relationship between measurements of the skin and subcutaneous fat and duration of the contraction ring. The figures presented in Table IV show that, in January, March and October, the men who exhibited marked myotatic irritability had, consistently, less subcutaneous tissue than those who were only slightly affected, and these in turn had less subcutaneous tissue than those who did not show the sign at all. The measurements were made with special calipers at the points specified. The same negative correlation between the duration of contraction and the thickness of the subcutaneous tissue held good for all four specific points at all three examinations. The proportions of men showing the contraction ring for four seconds or more was lower in October, 1949, than in January and March, viz. January 24·6 per cent., March 26·5 per cent. and October 12·5 per cent. For about four months before the October examination everyone had been getting an almost unrestricted intake of energy-yielding foods. These relationships appear to harmonize with the view that myotatic irritability is a manifestation of insufficient consumption of energy-yielding food. Marked myotatic responses were rarely seen in women, who were much fatter than the men, and seldom in children ; it was difficult to inflict an adequate stimulus without alarming the child.

TABLE IV

Relation between Thickness of Subcutaneous Tissues and duration of Contraction of Biceps in Seconds (Men)

Duration of contraction		0	1–3	4 or more
Number of Men...		30	80	36
January:	(1)	4·10	3·98	3·74
Subcutaneous tissue (mm.)	(2)	8·23	6·69	6·13
at site.	(3)	10·54	9·13	7·89
	(4)	10·46	9·44	9·17
Number of men		49	92	51
March:	(1)	4·26	3·89	3·40
Subcutaneous tissue (mm.)	(2)	7·21	6·62	5·50
at site.	(3)	10·12	9·23	7·68
	(4)	9·99	9·55	8·65
Number of Men...		66	80	21
October:	(1)	4·87	4·29	3·74
Subcutaneous tissue (mm.)	(2)	7·61	7·42	5·90
at site.	(3)	12·78	11·3	9·60
	(4)	12·07	10·6	9·20

The measurements were of the thickness of the double layer of subcutaneous tissues obtained by pinching the skin at (1) the arm over the middle of the biceps, (2) the arm over the middle of triceps, (3) the abdomen just below the 9th costal cartilage, (4) just below the angle of the scapula. The records refer to all men examined on the three occasions.

Tender Bones. In testing for oedema of the legs considerable tenderness was noted in some subjects, who would wince and might withdraw the limb. The findings showed slight but consistent improvement in children but no appreciable change in adults in the period March to October, 1949. In March 30 per cent. of children had " marked " bone tenderness, but only 18·5 per cent. in October ; the percentages for adults were 21 and 18·5 respectively.

The cause of this condition is uncertain, but there is no doubt that it is related to shortage of food, for it is rarely, if ever, found amongst well-fed populations, and is seldom observed in Great Britain. Although we do not know what food factors are involved, one would suspect a deficiency of calcium. To test this notion feeding tests were set going after the October, 1949, survey was finished, when a number of people still had tender bones. This enquiry was conducted by Dr. Borgmann, with help from Brigadier Cheyne, Public Health Adviser, British Sector, Berlin.

Twenty-four men and women showing the condition were divided into three groups and treated as follows:—

(A) 9 received one dose of 10 mg. of Vigantol and 0·5 litre milk daily ; Vigantol has a potency of 4,000 i.u. of vitamin D per mg. ;

(B) 7 had the same supplement as (A) plus 1·76 gm. daily of calcium as a salt of calcium ;

(C) 8 had a placebo daily.

The medical examination, which was done before, during and at the end of the feeding test, consisted of observations of bone tenderness, myotatic irritability, blood calcium, blood phosphatase and X-ray photographs of the legs before and after the period of feeding Vitamin D and calcium. Analysis of the findings showed no differences between groups A and B on the one hand and C on the other, and no change in any of these respects in A, B and C as the test proceeded. This test, therefore, threw no fresh light on the cause of bone tenderness. The persistence of the condition for over six months after the establishment of adequate feeding—during two of which months consumption of Vitamin D and calcium had been very abundant, shows that no dramatic relief of the condition is to be expected from good feeding.

Oedema. From November, 1948, until the blockade lifted, no new case of oedema was seen ; all subjects who showed it had had it before the blockade began, often as a result of a period spent in Russian detention camps. The incidence, in fact, fell between November and January ; probably the longer time spent in bed during the winter, because of fuel and lighting restrictions, was largely responsible for this lessened incidence, though the food position had, of course, improved. No change was seen between January and March, but paradoxically enough, in October, 1949, after the blockade had ended, there was a sharp rise in incidence.

In November, 1948, 6 men of the sample of 366 persons showed oedema of nutritional origin and 22 oedema due to other causes, such as varicose veins, cardiac conditions, etc.; in January the figures were 3 and 12 and in March the same. In October the same people showed oedema and, in addition, there were 10 new cases of nutritional and 7 of non-nutritional oedema. They had all increased in weight and all except 2 in fat covering, 10 so much so as to be beyond the range of our calipers. It is well-known that oedema may be precipitated by over-indulgence after privation, and this would seem to have been the case in our subjects. A contributing factor in bringing out a latent oedema was the increased exercise taken during the longer days of summer ; which was also presumably a factor in the increased incidence of oedema due to non-nutritional causes (e.g. varicose veins, heart disease).

Polyuria. This is a common accompaniment of famine conditions. It was a source of much inconvenience to the Channel Islanders during the period of German occupation (Banks and Magee, 1945). Most of the Islanders had to get up to urinate two or more times every night. The condition was attributed to the high proportion of water in the potatoes and other vegetables which formed such a large part of the ordinary diet. In Berlin during the blockade the staple foods—bread, macaroni-like preparations (Nährmittel), dried potatoes and vegetables, e.g. pulses, dried egg and milk, small rations of fats and canned meat or fish, contained relatively little water, although much may have been added in cooking, or drunk as soup or otherwise. Table V shows the percentage of persons affected in November, March and October, 1949.

TABLE V

Incidence of Nycturia in Berlin in 157 *Adults and* 209 *Children*

	No. of times of urination nightly	November	March	October
		Per cent.	Per cent.	Per cent.
Adults	0	26·8	24·2	40·8
	1–3	65·0	69·4	54·8
	4 or more	8·3	6·4	4·5
Children	0	47·4	33·0	51·7
	1–3	48·3	63·1	46·4
	4 or more	4·3	3·8	1·9

Bleeding Gums. Bleeding of the gums is one of the clinical signs of scurvy, but by itself it may mean nothing more than gingivitis due to an infection or irritation. During the blockade until January there was an increase in the number of cases. A feeding test with vitamin C showed no improvement whatever in the test group as compared with controls. Evidently, therefore, scurvy was not the cause of the condition. October, 1949, showed no significant improvement, though we got the impression that the gums had become firmer. The texture of the diet is an important factor in gingival health, and the mushy food may well have been the cause of the gingivitis.

Haemoglobin. In children and adults there was a fairly consistent rise in haemoglobin from November, 1948, through March to October, 1949. The mean values for 39 Tiergarten men were 15·42, 15·98 and 16·07 gm. Hb/100 c.c. in November, 1948, March and October, 1949, respectively; and for 48 Tiergarten boys 13·67, 14·32 and 14·82. The trend for the other groups was similar. This change is consistent with the changes in the nutritive value of the diet and with the improvements in weight, nutritional state and other criteria of health recorded in the surveys. The German physicians informed us that the values in October, 1949, for adults were about the same as the pre-war figures for healthy Germans and those for children in October seemed to be a little higher than for pre-war German children of the same age.

Serum Proteins. The values for serum protein for the whole period November, 1948, to October, 1949, remained constant at normal ranges. In six locality and occupation groups of 175 men and women the means were from 6·70 to 6·78 in November, 6·79 to 7·06 in March and 6·57 to 6·97 in October. The ranges for four groups of 196 children were very similar and both sets of observations showed that the lifting of the blockade and the improvement in diet had had no effect on the serum proteins.

Blood Pressure. Only in a single sample of 53 of the male industrial workers, was it possible to arrange for readings to be taken under constant conditions and by the same doctor on every occasion. The findings are given in Table VI. The changes are not statistically significant, but they are consistent with the changes in weight during the blockade and after. There is a small rise in the months immediately following the ration increase, followed by a steady period, and again followed by a rise after the blockade had lifted.

TABLE VI

Average Blood Pressure (mm. Hg.) of 53 Industrial Workers; Range and Standard Deviation

November, 1948		January, 1949		March, 1949		October, 1949	
Systolic	*Diastolic*	*Systolic*	*Diastolic*	*Systolic*	*Diastolic*	*Systolic*	*Diastolic*
126	78	129	76	129	76	131	81
90–180	50–100	85–190	45–105	100–190	45–105	105–185	70–95
15·6	9·3	20·6	11·0	17·2	10·4	14·8	5·7

In October, 1949, compared with November, 1948, 28 men showed increases in systolic blood pressure, 19 showed decreases and 6 were unchanged.

Endurance. Tests were started in January, 1949, but only 27 boys of ages ranging from 8-14 years were tested in January, March and October. The test consisted in ascertaining how long a child could hang on to a horizontal bar unsupported. In our studies the children were continuously encouraged and a good performance was rewarded with sweets ; the incentive was, therefore, a high one. It has been said that the test is too much affected by motivation (the will to do things) to be of value. Decline in the will to do things is amongst the earliest effects of under-feeding, and it is obviously important to get even an approximate measure of it. While a fall in endurance time might be due to deterioration in motivation or in physical efficiency, it is noteworthy that the average endurance time of these 27 boys rose steadily throughout the blockade to reach a level only a little below that of English boys of the same age (Milligan, 1947). Though the changes from one test to another are not statistically significant the findings like the growth records suggest a steady improvement, almost, but not quite, reaching English normal levels by October, 1949.

Time on bar in seconds

No. of children	January, 1929	March, 1949	October, 1949	English boys of same age October, 1949
27	115	123	140	150

Vitamin Deficiencies. No clinical evidence of vitamin A deficiency was seen either in the sample or in other subjects. The Crookes adaptometer was used to test 119 persons and gave values mainly within the normal range. The satisfactory position as regards vitamin A reserves was also confirmed by essentially normal values for vitamin A and carotene in sera which we despatched to Dr. H. M. Sinclair for test after the blockade had been lifted.

Every subject was tested for vibration sense; it was always present. Angular stomatitis of a very mild character was occasionally seen in the inmates of institutions, but never in the sample. There was, therefore, no clear evidence of deficiency of B vitamins.

Neither clinical rickets nor osteoporosis was observed in the sample and it was only after a long search that a very few cases of mild rickets were found in infant clinics.

Folliculosis. Between March and October, 1949, the incidence of folliculosis fell from 11·5 per cent. to 5·1 per cent. in adults, and from 35·9 per cent. to 17·7 per cent. in children. The significance of folliculosis is not clear and other factors, such as cold and lack of soap for washing may have affected its incidence, and we do not, therefore, attach nutritional importance to these findings.

Summary

1. In September, 1948, we visited Berlin to advise on ration scales during the Russian blockade. Rations were restricted in quantity and lacked variety. The proportions allotted to different groups were not related to their physiological needs. From our own observations we concluded that a significant proportion of the population was definitely under-nourished.

2. Improvements in the quantity and quality of the rations were advised, taking into account the limitations imposed by the air lift, together with some adjustment as between the different sections of the population. Most of these were adopted and were put into operation on 1st November, 1948. The improvement in diet amounted to an overall increase of some 200 calories per head daily.

3. At the same time a systematic study was begun of the nutrition of a selected sample of the populace. This was repeated at intervals until four months after the blockade was lifted.

4. During the blockade, moderate improvements were recorded in weight, general nutritional status, myotatic irritability, haemoglobin levels, capacity for physical endurance, and blood pressure. Tenderness of leg bones showed little change, and the incidence of bleeding gums increased slightly.

5. After the blockade, further improvements were recorded in the incidence of all the signs observed, except of bleeding gums; even in this case some increase in firmness of the gums was noted.

6. No new cases of nutritional oedema developed during the blockade. (The condition was confined to men, especially those who had been in Russian detention camps). The severity decreased from November to January, probably because of the enforced rest during the long nights without lighting, but the condition became worse again in spring, and especially after the end of the blockade. Improved feeding after a period of want, and increased exercise, are therefore evidently important factors in nutritional oedema. Oedema attributable primarily to circulatory and renal causes was observed more often than oedema of strictly nutritional origin.

7. Tests were carried out to ascertain the causes of some of the signs observed. Myotatic irritability did not respond to treatment with vitamin Bl (as had been observed elsewhere) but was found to be closely related to under-nutrition. It probably originates in the muscle fibres and not in the nerves or neuro-muscular junction. Bleeding gums did not respond to vitamin C, and the condition was ascribed to the mushy state of the food. No improvement was found in tenderness of bones following the administration of calcium and vitamin D.

8. No evidence of deficiency of vitamins A, B or C was found; the investigation included therapeutic tests. A very few cases of mild rickets were found in infant clinics after prolonged search; no other evidence of vitamin deficiency was seen.

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A HALF-WAY HOUSE FOR THE AGED SICK: AN EXPERIMENT IN SOCIAL MEDICINE

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In the days of Sydenham, the common medical problems were the acute diseases and the fevers which took up about two-thirds of a physician's time. Chronic diseases were unimportant by comparison. But the great improvements in sanitation and the introduction of sulphonamides, penicillin, streptomycin and so on, entitle us to say with Molière: " Nous avons changé tout cela! " Our difficulties to-day arise from the long-term maladies of an ageing population. The present hospital system of this country, which has grown to meet the needs of acute or sub-acute illness in younger persons, is not easy to adjust to the new circumstances. The numbers of the elderly chronic sick have increased, are still increasing and will not diminish during the next twenty years. Consultants in general hospitals complain that patients of this type block beds badly needed for the treatment of more urgent cases. General practitioners state that it is difficult for them to get an elderly person admitted without a long wait, during which deterioration is inevitable. This creates a vicious circle through which more and more chronic sick get less and less treatment. So much is common knowledge. Let us examine some of the measures taken to improve the situation.

One step, already taken, is to ask general hospitals to admit a proportion of aged or long-term patients. As we have already seen, this is not satisfactory. The former statutory outlet to the municipal hospitals no longer exists, so that transfers are not easy to arrange. Another step has been the organisation of " Geriatric Units " to cope with the needs of the elderly sick. These vary considerably in size and activity. A large unit with an experienced physician in charge and adequate staff (both medical and nursing) working together as a team, will get extremely good results. Perhaps the best-known example of this is the West Middlesex Hospital, where Marjory Warren restores about two-fifths of her hemiplegic patients to a level of moderate activity. Even a small unit, like that at St. Helier, Carshalton, can cope with 1,000 cases in three years by means of domiciliary visiting on a large scale. My own beds at St. John's Hospital, Battersea, have increased their turnover fourfold during the past three years by a combination of domiciliary visiting with busy out-patient clinics. But sooner or later, we all meet the same twin problems. One large group of our patients reaches a state in which they can no longer respond to treatment and must be looked after for the rest of their lives. Ideally, these should go to a " long stay annexe ", where general nursing care and supervision were available outside active hospital wards. In practice, the shortage of nurses makes this impossible. Another group recover enough to live sheltered lives, but not enough to return home as completely cured. These " frail ambulant " patients are mainly rehabilitated hemiplegics, arthritics, bronchitics or mild senile dements, who should be kept under medical observation to prevent subsequent relapse. These are the patients who can be sent to a " Half-way House " or Home for the Aged Sick, thus clearing badly needed hospital beds.

This idea of accommodation for the frail ambulant was first discussed in medical circles about five years ago. It originated, in part, from the practice of Cosin at Orsett Lodge Hospital, and, in part, from my own experience at the Royal Hospital, Chelsea, during the war. We found that partly rehabilitated patients could live quite happily in dormitory accommodation under supervision, without need for a full nursing staff in attendance. The suggestion has been taken up by King Edward's Hospital Fund for London and by the National Corporation for the Care of Old People. With the support of these two bodies a small number of homes have been set up already. One of these is working in conjunction with the Geriatric Unit at St. John's Hospital, Battersea, to serve the Battersea and Putney group of hospitals. It has already served a useful purpose in relieving pressure by taking a number of frail ambulant and partly recovered elderly patients who would otherwise have occupied beds needed for active treatment. Several lessons have been learnt about the use of this type of accommodation, which are recorded here for the benefit of those who may be interested.

The Half-way House in question has room for 24 patients and three resident staff besides the matron, who has a small cottage in the grounds. For this and other reasons, we do not have any nurses on duty at night. Hence patients who need much constant attention are not accepted. The atmosphere is that of a home, not of a hospital. There are bedrooms and sitting-rooms, not wards and day-rooms. The residents are encouraged to take part in the housework. Some wash the dishes, others peel potatoes, dust, or help the cook. They are allowed to walk to the village at Roehampton if they are able—about two hundred yards away. Several of them find an interest in the garden. Since some of the first cases admitted had spent the past five or ten years in hospital, this was a great change for them. As a result, most of them improved and some have since become fit for discharge, either to their own home or to hostel accommodation.

Of course, a certain number have been found unsuitable for this home. Having only a small nursing staff and limited facilities for treatment, it has been our practice to transfer back to hospital all patients needing much medical or nursing attention. For example, an old lady with an exacerbation of pain from her gastric ulcer was re-admitted to hospital; as were several old people who have had severe falls and needed an X-ray; and two residents who have developed cardiac failure. On the other hand, we have found that most of the ambulant hemiplegic cases have improved very considerably in the home. All the patients with arthritis are more active and complain less of their aches and pains. A number of old people with slight confusional states do much better in community life than just sitting in a hospital ward. We have also admitted one or two patients from my out-patient clinic to avoid the need for admission to hospital. In this way, the turn-over in the beds of the parent geriatric unit has been increased considerably. It will be obvious that a close link between the half-way house and the geriatric unit is essential. In some of the larger homes, which boast nurses on night duty, it may be possible to allow a certain number of patients to be in bed. But there is a danger of such a home thus becoming a long-stay annexe for the irremediable chronic sick instead of a true half-way house. If the emphasis is placed on activity and occupation, it is possible to have a steady turn-over of cases. Close co-operation with voluntary and statuary hostels for the aged will facilitate this. It may be possible to exchange an active rehabilitated patient for one who can be treated successfully in the geriatric unit, when transferred from hostel accommodation. This two-way traffic has advantages from several points of view. But it is only possible when there is a proper geriatric unit giving skilled treatment and reablement to a number of long-term elderly sick. Continuity of medical supervision is essential—the whole system breaks down without it. It must be easy to transfer patients to and from the half-way house with a minimum of delay, or grave nursing and administrative difficulties may result.

The financial arrangements of this type of homes vary from one to another. Generally speaking, the Regional Board pay an agreed weekly sum per occupied bed for maintenance. This is about two-thirds of the amount which a patient costs in a chronic hospital, on the average. The building remains the property of the parent body organising the home. In practice it is found that three-quarters of the expenses are taken up with wages, salaries, rent, rates, taxes, heating and lighting charges. Food, drugs and dressings make up the rest. Since it seems to cost about as much to keep two patients in hospital as three in a half-way house, there are financial advantages in this scheme. These are enhanced when a steady turn-over of residents relieves the congestion in hospital beds and thus allows more patients to be admitted from the waiting lists. But if there is little or no flow of patients through the home, it becomes merely a cheaper form of chronic nursing home and a doubtful investment. However, this scheme is only in its infancy and will doubtless develop much in the future. It marks a definite step towards solving the problem of the chronic aged sick, and is worthy of further expansion.

Summary

An account is given of the experience gained in running a " Half-way House for the Aged Sick ", and its effect upon the hospital waiting list.

It is emphasised that close contact must be maintained with an active geriatric unit to obtain the best results.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

JULY, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

	July 7	July 14	July 21	July 28	Average weekly figures for July, 1950
Scarlet Fever	816	805	830	758	1,096
Whooping Cough	3,358	3,279	3,380	3,261	3,914
Diphtheria	22	35	30	36	50
Measles, excluding Rubella ...	8,544	7,666	6,306	5,258	8,839
Acute Pneumonia	358	348	292	308	293
Meningococcal Infection ...	39	36	37	35	33
Acute Poliomyelitis (Paralytic)...	40	50	61	50	170
,, ,, (Non-paralytic)	60	70	73	71	78
Ophthalmia Neonatorum ...	40	38	47	27	34
Puerperal Pyrexia and Puerperal Sepsis	73	92	89	114	80
Dysentery	368	292	301	226	208
Paratyphoid	23	29	37	53	9
Typhoid	7	4	5	8	5
Smallpox	—	—	—	—	—

No cases of Cholera or Plague.
1 case of Typhus Fever (not louse born) in the week ended 14th July.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Dorchester Laboratory

The Associated laboratory at Glyde Path Road, Dorchester, Dorset, has now been taken over as a Constituent unit of the Public Health Laboratory Service (*Tel.:* Dorchester 864). Dr. G. H. Tee has been appointed Director.

Wakefield Laboratory

Brigadier H. T. Findlay has taken over the direction of the Wakefield laboratory from Colonel W. F. Lane, who has been appointed Director of the new laboratory at Bedford. The date of opening of the Bedford laboratory will be announced later.

AN OUTBREAK DUE TO *Salmonella wien*

J. H. Wildman, M.R.C.S., D.P.H., Medical Officer of Health, E. Herts Combined Sanitary Districts,

C. Grant Nicol, M.B., D.P.H., Barrister-at-Law, a Medical Officer of the Ministry of Health, and

G. H. Tee, M.A., M.R.C.S., Public Health Laboratory Service, Cambridge.

Over the week-end 2nd-4th September, 1950, 49 residents in the adjoining towns of Broxbourne and Hoddesdon developed diarrhoea and vomiting after eating tinned ham imported from France. One tin of this ham had been opened at 9 a.m. on 2nd September in a grocer's shop, the ham being freshly sliced for each purchaser. The time between ingestion and onset of symptoms varied from 4 to 59 hours. Initial laboratory reports suggested that the infection was due to *Salmonella paratyphi B*; this organism was reported to have been isolated from the actual substance of the ham, and from the stools examined from every one of the consumers who had developed gastro-enteritis. Pending the phage-typing of the cultures and confirmation of the regional laboratory findings, the position was that 59 persons had eaten infected ham, and 49 of these were ill with an initial gastro-enteritis.

Of these 49 patients, 47 had gastro-enteritis only and in 2 this merged into an enteric-like illness. In addition, there were 2 persons at risk (with positive stools) who had no initial gastro-enteritis but who developed clinical enteric-like illness after incubation periods of five and seven days respectively. Eight symptomless excreters were detected. Of the 59 persons who had eaten the infected ham 57 could be examined and the organism was isolated from the faeces of all of them. The other two persons had already left the district.

Control Measures

On 8th September when an initial appreciation of the position was made, it was felt that immediate action must be taken to minimize the potential danger of cases of paratyphoid fever arising among those at risk. With the co-operation of the County Health Department at Hertford, arrangements were made for all these persons to be visited at first twice daily, then once daily, by a nurse who took and recorded the temperature, asked about general health and instructed them to provide a daily specimen of faeces for laboratory examination. The nurses met the district medical officer of health at a daily consultation, and all residents at risk remained under the closest supervision by their medical practitioners and by the medical officer of health.

It was also decided that every patient should be given a short course of chloromycetin (chloramphenicol, Parke, Davis and Co.). A meeting was arranged with the local practitioners to whom the position was explained. It was agreed (1) that one half of the patients at risk—those most recently infected—should receive four 250 mgm. capsules of chloramphenicol every six hours for adults and proportionately less for children, for three days, beginning on the 9th September ; and (2) that the other half—those with the earliest infection—should receive a similar course of chloramphenicol commencing on the 12th September, i.e. after an interval of three days. It was also agreed that any patient in this second group whose clinical condition was unusually severe should receive chloramphenicol without delay. Thus all patients were to be offered chloramphenicol and of these 49 in fact were treated with it. Six persons declined treatment, 3 had already left the district, and one died of pre-existing chronic illness.

Table 1 shows clearance times of *Salmonella* from the faeces of 49 treated and 3 untreated patients. No significant difference was apparent in the time taken by the members of any of these groups to give consistently negative bacteriological results.

TABLE 1

Duration of excretion of Salm. wien *in untreated patients and in patients treated with chloramphenicol*

Number of patients	1	1*	4*	20	2	6	12*	1*	1	1	3
Interval in days between infection and starting course of chloramphenicol.	3	4	6	7	8	9	10	11	12	13	Received no chloramphenicol
Interval in days between† starting course of chloramphenicol and faecal culture being consistently negative.	30	22	16 73	11 73	3 43	3 41	3 48	N.K.	14	N.K.	30 58

* One patient in each of these groups developed an enteric-like illness.

† The earliest and latest days of clearance are indicated by the upper and lower figures respectively.

N.K. Not known.

Laboratory Findings

The first specimens (two samples of ham, one ham sandwich, one specimen of vomit and five specimens of faeces) were received by the Public Health Laboratory at Cambridge on 5th September, and on the following day it was reported that from all these specimens there had been isolated an organism of the salmonella group. A visit to the grocer's shop at Broxbourne, where the ham had been sold on 2nd September, was made at noon on this day by the medical officer of health and the chief sanitary inspector together with workers from the laboratory at Cambridge. Here cultures were made from various parts of the bacon-slicing machine used for cutting the ham and from a marble slab upon the counter where the slices had been placed. A fly was also taken for examination. From none of these cultures was a salmonella isolated and it was therefore considered that there was no danger arising from the continued use of the shop for the sale of bacon, cheese and butter provided food handlers were free from infection. As a precaution it was advised that the counter be well scrubbed with hot water and soap before swabbing it over with a hypochlorite solution.

It was arranged that, as far as possible, daily specimens of faeces should be examined and, for this purpose, specimens were sent by train to Cambridge where they were cultured on the same day.

Cultural Methods

Each specimen was cultured upon Wilson and Blair's medium (Glass and Tabet, 1938) and Leifson's medium (Hynes, 1942); a tube of tetrathionate broth (Rolfe, 1946) was also inoculated. Plates were examined after overnight incubation at 37°C. Cultures on Wilson and Blair's medium, if negative, were then reincubated for a further 24 hours, after which a second examination was made. Tetrathionate broths were subcultured after 24 hours' incubation on to Leifson's and Wilson and Blair's media.

Results

It was soon evident that the use of Wilson and Blair's medium was of great value in obtaining quick results. Out of the first 100 positive specimens examined:—

100 were positive on Wilson and Blair's medium, of which 86 were positive at the 24-hour reading; 14 were positive after further incubation.

71 were positive on Leifson's medium.

99 were found positive on subculture of the tetrathionate broth.

If, therefore, only Leifson's medium had been used 29 per cent. of positives would have been missed. Had tetrathionate broth alone been used 99 per cent. of positives would have been found, but with a delay of an additional day in reporting them.

Cultures from the piece of ham remaining unsold in the grocer's shop showed the presence of the organism on the surface (together with a few *Staph. albus*) and also in the muscle. Cultures of the fat were sterile. Distribution of the salmonella within the muscle was evidently not uniform since a portion excised with the intention of making a bacterial count proved sterile. The organism was isolated from portions of the purchased ham seized by the sanitary inspector, including a ham sandwich. It was also isolated from the faeces of each of the 57 local residents known to have eaten the ham—from 55 of them at the first attempt and from 2 at the second. Owing to the ease with which the organism could be isolated from the faeces, urinary cultures were not considered necessary.

It is, therefore, reasonable to suppose that the ham was heavily infected before the tin was opened in the grocer's shop, indicating that the heat treatment to which the tinned ham was exposed at the place of canning was insufficient. This evidence is supported by the fact that other tins from the same batch were bacteriologically unsatisfactory.

The value of early culture of faeces of all those known to be at risk is emphasized as being of great value in controlling this outbreak. It enabled positive food handlers to be detected and suspended from work as soon as possible.

It is also notable that two persons were found to have positive faeces when the first culture was made 5 and 7 days respectively before they developed symptoms—precocious carriers. One of these two cases might have been considered to be a secondary case if early cultures had not been made.

Treatment with chloramphenicol made no difference to the colonial morphology or to the proportion of positives isolated on the two solid media used. Tested *in vitro* the strain was found to be sensitive to chloramphenicol to the same degree as stock strains of *Salm. paratyphi B* and *Salm. enteritidis*.

Figure 1 shows the proportion of cases found positive or negative at weekly intervals for eight weeks. The conventional "three consecutive negatives" provided no evidence of freedom from infection except when the interval between each was at least one week.

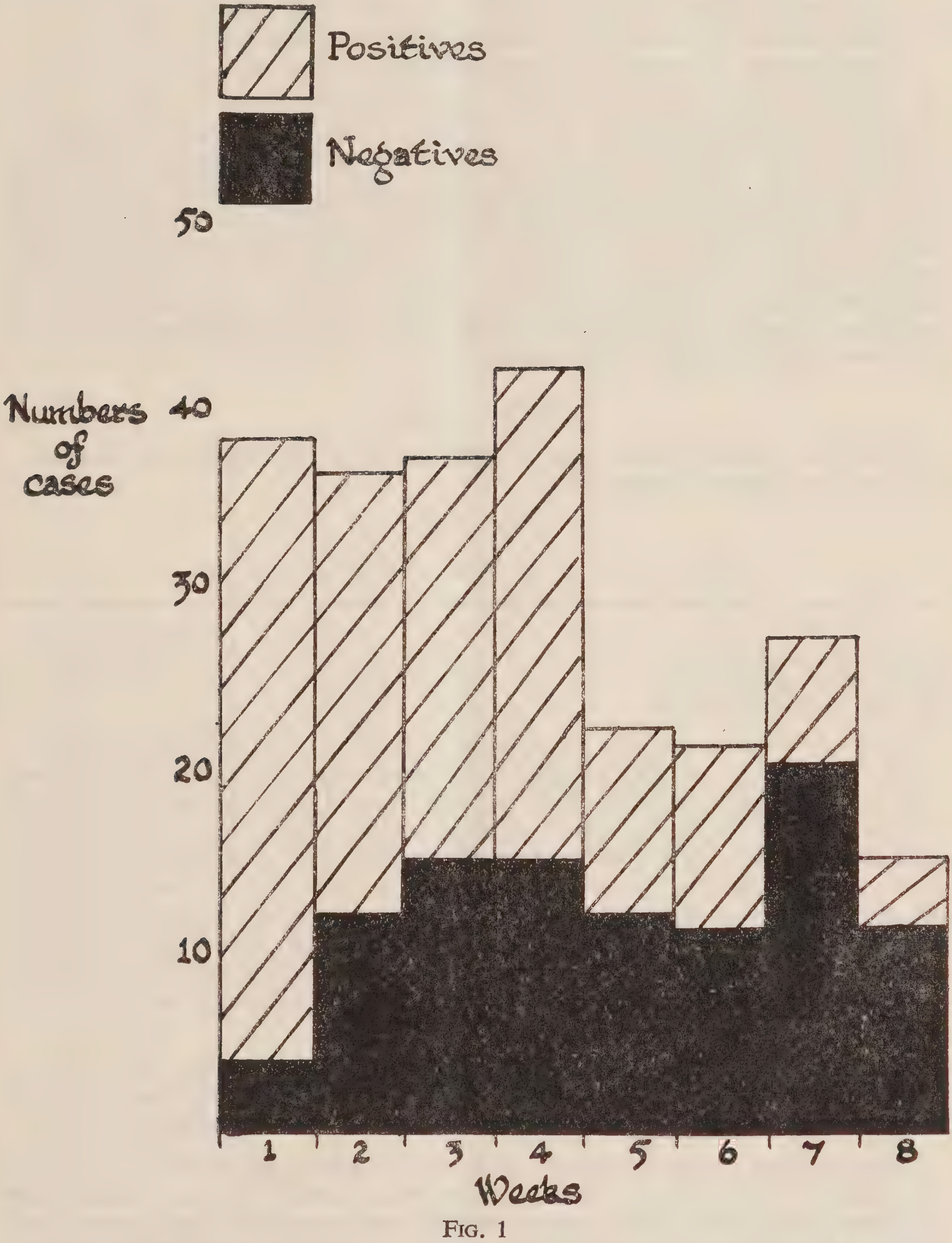

FIG. 1

Proportion of positive and negative bacteriological findings in patients examined during successive weeks.

Investigation of organisms

Morphologically positive colonies were subcultured on to MacConkey's agar and thence inoculated into sets of sugars, including glucose, mannitol, maltose and dulcitol, which were fermented with the production of acid and gas; lactose, sucrose and salicin were not fermented. Indole was not produced.

Tube agglutinations in the water-bath at 52°C. were carried out using formolized suspensions for the H and boiled suspensions for the O antigens. Sera used were supplied by the Standards Laboratory, Colindale. The titres were:—

Salmonella paratyphi B H 1/250.
Salmonella paratyphi B O 1/250.
Non-specific Salmonella I (containing 1, 5) 1/250.
Non-specific Salmonella II (containing e, n, x), nil.
Salmonella typhi-murium H, nil.

It was therefore considered that this organism was *Salmonella paratyphi B.* One subculture of all strains isolated was sent to the Salmonella Reference Laboratory where it was examined by Dr. Joan Taylor. All of these proved to be identical, having the antigenic structure iv, xii : b—l, w, a constitution not previously found in this country. The provisional designation *Salmonella broxbourne* was suggested; but it was learned later by Dr. Taylor that an organism with a similar antigenic structure (*Salmonella wien*) had been described recently in Vienna (Roschka, 1950), and comparison of the two organisms showed them to be identical.

Clinical Findings

From the clinical aspect the 59 persons who ate infected ham can be grouped as follows (Table 2):—

TABLE 2

Distribution of the clinical types of disease

		Incubation period
Acute gastro-enteritis only	47 cases	4–16 hours
Clinical paratyphoid fever	4 cases	1–15 days
No symptoms (excreters only)	8 persons	—

The sufferers from simple acute gastro-enteritis formed the largest group and numbered 47 persons. They all ate infected ham during the 2nd and 3rd September, 1950, and the majority developed diarrhoea and vomiting within two days. One child vomited after only four hours and 38 persons after intervals varying from eight to twenty-seven hours; 7 adults became ill after periods ranging from twenty-eight to forty-six hours after eating the ham. Finally, one woman of 64 years of age who had experienced no previous symptoms of gastro-enteritis developed diarrhoea and a temperature of 100° F. thirteen days after eating the ham, although her stools were positive for *Salmonella wien* from the outset.

Three of the patients were treated in hospital, 3 others left the area on the day after eating the ham, and the remaining 41 patients were visited daily at their homes in order to observe their clinical progress.

The smallest group consisted of 4 adults only, who were diagnosed *in hospital* as mild cases of clinical paratyphoid fever. Three patients were ill from the outset with gastro-enteritis which merged into paratyphoid fever. On the tenth day of illness, 2 of these patients developed rose spots. The final patient to be admitted to hospital was a woman of seventy who had suffered no initial illness although her stools were positive seven days previously. Fifteen days after eating some of the ham she suddenly developed diarrhoea and vomiting, associated with a temperature of over 101° F. but no rash. All 4 patients gave a positive Widal reaction and continued to excrete *Salmonella wien* in their stools for several weeks.

Eight persons at risk developed no symptoms whatsoever although they continued to excrete *Salm. wien* for periods ranging from two to eight weeks after infection.

The age and sex distribution did not reveal anything of significance in any of the three groups of patients shown in Table 2.

Incubation Periods

The period between the eating of infected ham and development of the first symptoms (diarrhoea or vomiting) varied between 4 hours and 59 hours.

Sartwell (1950) states that the usual frequency curve of incubation time has the form of a logarithmic normal curve. Our own figures being unusually accurate—the incubation periods were known to the nearest hour in 49 instances—we followed Sartwell's procedure in studying the distributions. Frequencies were grouped in time intervals (hours) ; the cumulative frequencies and their cumulative percentages were found ; the time intervals were converted to logarithms. These results were plotted upon normal probability paper, the ordinate representing cumulative percentages of cases, the abscissa showing the time scale in logarithms. Figure 2 shows that the resulting curve approximates closely to the fitted line, with the exception of points representing unusually short or unusually long incubation periods.

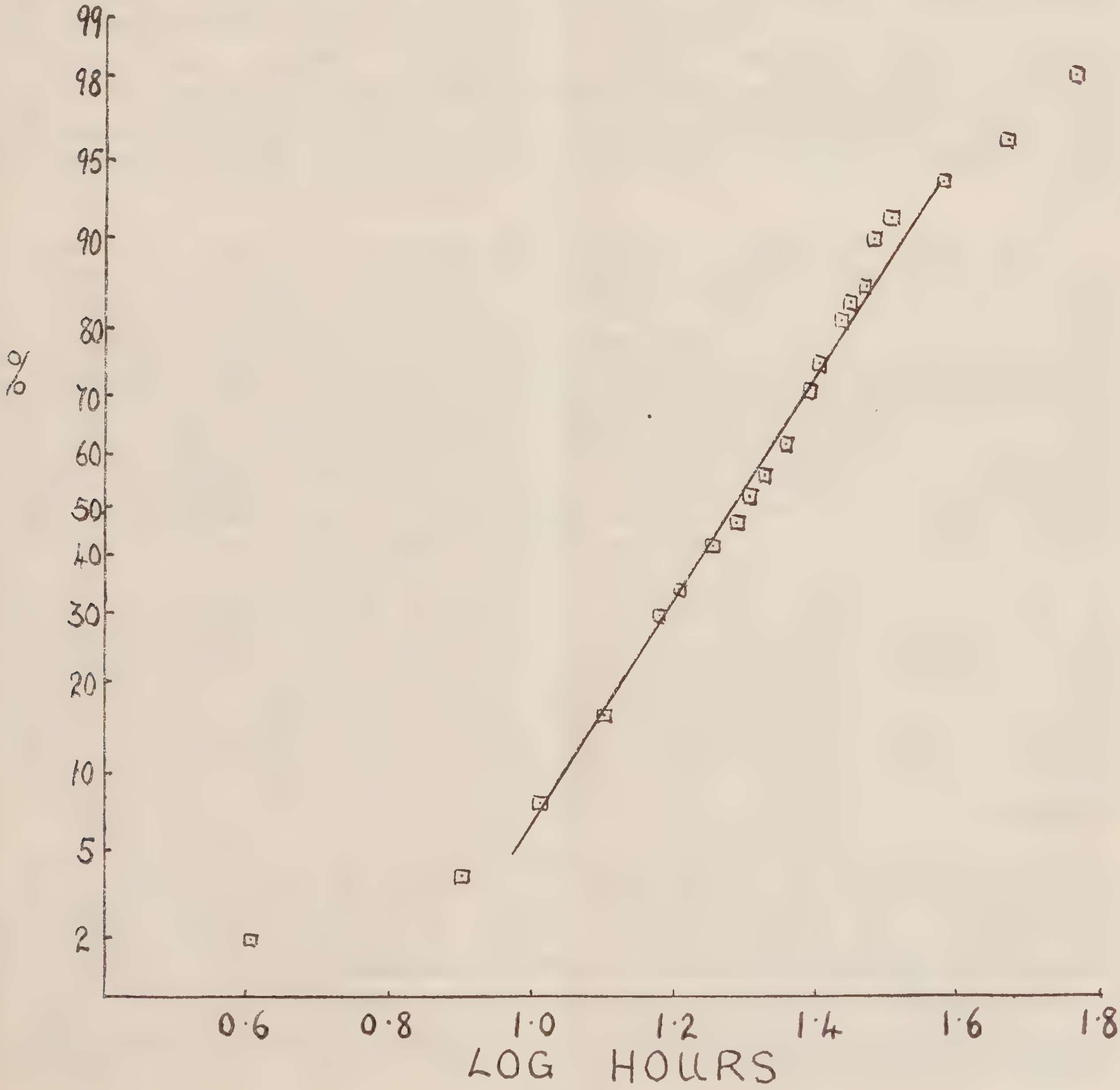

FIG. 2
Frequency distribution of incubation periods.

Administrative Aspects

In addition to the clinical and bacteriological features of the outbreak already described, the coincident administrative action presented certain interesting problems. The unsold portion (12 lb.) of the offending ham together with its accompanying tin and the remaining stock of this brand of ham (one unopened blown tin and one unopened sound tin) were surrendered voluntarily by the grocer. The causative salmonella was isolated in almost pure culture from the remainder of the ham, and from portions of ham recovered from purchasers, but not from the inside surface of the containing tin or from either of the other tins of ham and their contents. Inquiries were then made as to the possibility of there being further infected tins of ham in other districts. It was found that the three tins of ham in Broxbourne were part of an original importation from France of 40 cases (240 tins). The distribution of this consignment was traced to provision and cooked food shops over a wide area in London and the Home Counties, including some 20 different local authorities. Of this ham, some had been sold and eaten without any reported ill-effect; some remained unsold; and a number of tins had been surrendered as " blown " and apparently unsound. The sanitary authorities concerned had submitted nine further tins and an informal sample from a tenth tin for examination at various laboratories of the P.H.L.S. but in no case was either *Salm. wien* or *Salm. paratyphi B* isolated; in fact no further *Salmonella wien* infection was discovered despite a widespread examination of the remaining tins of this brand. From the tins examined certain of the cultures yielded coliform bacilli, aerobic spore bearers, non-haemolytic streptococci, and *Proteus;* others gave entirely negative results. Although the specific disease-producing organism was not found, the consignment was bacteriologically unsatisfactory and action was taken by the Ministry of Food.

Of the 59 persons affected, 4 were food handlers—the grocer from whose shop the ham was sold, his assistant, the owner of a fruit and vegetable shop, and a café waitress. They all suffered an initial gastro-enteritis, and were found to be excreting salmonellae of the type found in the ham. The grocer's assistant, the greengrocer and the waitress were incapacitated by their illness and the question of their exclusion from work did not therefore arise. In the grocer himself the illness had been very mild, and he was able to continue work. It was found advisable therefore to apply the provision of the Public Health (Infectious Diseases) Regulations, 1927, and to serve a formal notice prohibiting him from remaining at work as being a carrier of enteric fever. This decision was later reviewed in the light of the final laboratory findings, but after consideration of all the factors and, in particular, that the disease had been clinically an enteric fever in certain of the cases, it was decided to adhere to the original decision, and to exclude him from work requiring the handling of food. The statutory notice remained in force for over four weeks and was finally withdrawn after stool specimens had remained consistently negative.

Of the 59 persons at risk 2 moved out of the district, and 1 was temporarily absent from the area. These immediately came under the surveillance of the medical officer of health of their new locality. It is of interest that a large proportion of those so examined remained persistently or intermittently positive for as long as nine weeks, yet there were only 3 secondary cases in persons who had not eaten the infected ham, all close contacts of infected families.

Some Other Unusual Salmonella Outbreaks

There are several instances in the recent literature of other rare salmonella types associated with enteric-like infections, none of which occurred in this country. Of these, the following are the most notable examples.

Salm. blegdam. Fenner and Jackson (1946) described 50 cases of enteric-like infection among soldiers in New Guinea. This organism had been previously isolated by Atkinson and her colleagues (1944) from 4 similar cases in the same country and was later reported by Holt and Newton (1948) in Louisiana.

Salm. sendai was isolated from 8 patients with symptoms of enteric fever by Dunlop (1950).

Although it is widely known that many salmonellae usually causing acute gastro-enteritis can occasionally produce an enteric-like disease, the converse of this is also found occasionally ; that is, a salmonella usually causing enteric symptoms may produce an illness with a shorter incubation period presenting as an acute gastro-enteritis which may or may not be followed by enteric symptoms. Such an outbreak was described by Warren (1941) in which, out of 64 persons who ate portions of a trifle infected with *Salmonella paratyphi B,* 43 had diarrhoea and vomiting within seventy-two hours and 9 had these symptoms only but no subsequent enteric illness.

Anderson (1940) recorded that in an outbreak of *Salmonella paratyphi B* in Glasgow with 118 clinical cases, 59 presented as acute vomiting and 48 had diarrhoea at the onset of illness.

It has been noted by Edwards and Bruner (1943) and by a number of other workers that *Salm.cholerae-suis* is specially liable to give rise to invasive manifestations. Illness of " continued fever " type has also been noted in infections with *Salm. dublin* and with the *chaco* variety of *Salm. enteritidis* (Savino and Menéndez, 1934).

Summary

An outbreak of illness following the consumption of imported tinned cooked ham is reported.

From the ham, and from the faeces of each of 57 of those examined who were known to have eaten the ham, there was isolated an identical salmonella of a type hitherto unreported in this country (*Salmonella wien*).

The incubation period varied between 4 hours and 15 days. Out of 59 consumers at risk:—

47 developed acute gastro-enteritis only.
4 developed clinical paratyphoid fever.
8 were symptomless excreters.

In addition to these cases, 3 secondary cases of gastro-enteritis occurred from whom the same type of *Salmonella* was also isolated.

Two patients had positive faeces 5 and 7 days respectively before onset of any illness—precocious carriers.

Three consecutive negative cultures of faeces provided evidence of freedom from infection only when taken at weekly intervals.

Analysis of the incubation periods showed that they conformed to the logarithmic normal curve.

We wish to acknowledge with grateful thanks the help, advice, and co-operation given by Dr. W. H. Bradley of the Epidemiological Division of the Ministry of Health ; by the County Medical Officer and staff of Hertfordshire County Council, without whose co-operation the close follow-up

of all the cases would have been impossible; by our general practitioner and hospital colleagues; by Dr. C. H. Jellard, Dr. F. M. Mackay-Scollay and Mr. V. Rolfe, F.I.M.L.T., for invaluable technical assistance; and by Miss Hopkins of Cambridge in the tabulation of laboratory findings.

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SALMONELLA WIEN

S. Hilda Douglas, M.Sc., and Joan Taylor, B.Sc., M.B., D.P.H.,
Salmonella Reference Laboratory, Colindale

In September, 1950, 38 strains of an organism believed to be a salmonella were received for identification. These strains were isolated from the following material:—

3—ham.
1—ham sandwich.
1—vomitus.
32—faeces from 29 patients.
1—urine.

Three strains, one each from ham, faeces and vomit were studied in detail. All strains were motile and produced colonies typical of the salmonella group. Acid and gas were formed from glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose and trehalose. Acid was formed in dextrin after 5 days' incubation. No change was produced in lactose, sucrose, salicin, adonitol, inulin, raffinose, or inositol. The Voges-Proskauer test was negative and the methyl-red positive; urease and indole were not formed, and gelatin was not liquefied. There was growth on Simmons citrate agar and H_2S was produced.

On serological examination these strains were found to have the somatic antigenic structure IV, XII. The strains were diphasic, both phases being isolated from the majority of primary cultures. The flagellar antigens were b and l,w. Of the 38 strains investigated 35 were mixed Phases 1 and 2, and three appeared to be in Phase 1, the second phase being isolated only after culture in weak agar to which *Salm. paratyphi B* H (b) serum had been added. It was shown by reciprocal absorption tests that the somatic antigen of these strains was identical with that of *Salm. essen*; that flagellar Phase 1 was identical with that of *Salm. paratyphi B* H (b); and that flagellar Phase 2 was identical with *Salm. dar-es-salaam* H (l,w).

In July, 1950, Dr. Roschka described a new salmonella type at a meeting of the Oesterreichische Mikrobiologen-Gesellschaft in Salzburg. Dr. Kauffmann kindly supplied a strain of this new type named *Salm. wien* by Kauffmann ex Roschka and Dosch (Kauffmann, 1951). *Salm. wien* has the antigenic structure IV,XII: b—l,w. Absorption tests showed that *Salm.*

wien and the three strains isolated from the Broxbourne outbreak were antigenically identical. In addition the biochemical reactions of *Salm. wien* and of the three strains tested were identical.

In a group of 14 persons examined there were 13 with symptoms of a mild gastro-enteritis and 1 without any sign of illness. *Salm. wien* was isolated from 5 of the patients and from the symptomless excreter. Serum from all members of this group was taken about four weeks after the start of the outbreak and tested for specific agglutinins against pure suspensions of factors IV,XII : b, and l,w with results shown in Table 1.

Serum from the symptomless excreter failed to agglutinate O and H suspensions of the organism. The highest titre was obtained from a patient who suffered from diarrhoea and vomiting; her serum agglutinated O, H Phase 1 and H Phase 2 suspensions of the organism to titres of 1/40, 1/2,560 and 1/40 respectively.

TABLE 1

Human sera agglutinating O and H suspensions of Salm. wien *at different titres*

Agglutinating titre of sera	Number of sera positive		
	0 = IV, XII	H (b)	H (1, w)
< 1/20	4	2	8
1/20–1/80	8	7	4
1/160–1/320	2	3	2
>1/320	0	2	0

Discussion

All strains of *Salm. wien* isolated from the outbreak described, being mainly in Phase 1, were agglutinated to titre by *Salm. paratyphi B* H (b) serum. As *Salm. paratyphi B* infection is not uncommon in this country, and as many patients suffered from a clinical disease compatible with the diagnosis of paratyphoid fever, it was at first thought that the outbreak was due to infection with *Salm. paratyphi B*. The full serological investigation of strains isolated showed that the organism differed from *Salm. paratyphi B* in having the l,w antigens in the second phase instead of 1,2. This outbreak of *Salm. wien* infection is the first to be recorded; two sporadic cases have been published, the first by Dr. Roschka in Salzburg, and the second in Montreal. The organism from the latter case was named *Salm. montreal* by Laidley *et al.* (1951) but was later found to be identical with *Salm. wien*.

Summary

From an outbreak of food poisoning 38 strains of *Salm. wien* were isolated, 4 from infected food, 34 from patients.

We should like to express our acknowledgment of the technical assistance rendered by Mr. D. C. Miller.

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A NEW SALMONELLA TYPE, *Salmonella oxford,* ISOLATED FROM A GUINEA-PIG

W. H. H. Jebb, M.A., M.D., Public Health Laboratory, Oxford, S. Hilda Douglas, M.Sc., and Joan Taylor, B.Sc., M.B., D.P.H., Salmonella Reference Laboratory, Colindale.

A guinea-pig, one of the normal stock animals, was observed to be in poor condition and to be suffering from diarrhoea. None of the other stock guinea-pigs was affected. The sick animal was killed and cultures were made from the contents of the gut. Primary cultures on deoxycholate citrate agar and Wilson and Blair's medium showed numerous colonies typical of the salmonella group of organisms.

Bacteriology

The organism proved to be a Gram-negative motile bacillus which fermented, with the production of acid and gas, glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose, trehalose and inositol. A small amount of acid was produced in dextrin. It failed to ferment lactose, sucrose, salicin, adonitol, inulin and raffinose within three weeks. Growth occurred on Simmons citrate agar ; hydrogen sulphide was produced but indole was not formed ; urea was not hydrolysed and there was no liquefaction of gelatin. The Voges-Proskauer test was negative and the methyl-red test positive.

On serological examination this organism appeared to have the antigenic formula III, X : a — 1, 7. When tested with absorbed sera for factors 2, 3, 5, 6, 7, it was agglutinated by sera for factors 2, 3 and 7. Single colonies of each phase were isolated from the original culture. The organism was agglutinated to titre by *Salm. anatum* O serum. Absorption of this serum by *Salm. anatum* completely removed the homologous agglutinins ; similarly *Salm. anatum* O was agglutinated to titre and removed all somatic agglutinins from an antiserum prepared with the organism. Therefore the somatic antigenic structure is represented by the symbols III, X.

H Phase 1 of this organism was agglutinated to titre (1/25,600) by *Salm. paratyphi A* H serum. Absorption of *Salm. paratyphi A* H serum with the new type left a residual titre of 1/50 (0·2 per cent.). This residuum is accepted as indicative of a minor antigenic difference. A serum made with the new type agglutinated to titre *Salm. paratyphi A* H. Absorption of serum made with the new type by *Salm. paratyphi A* H left a residual titre of 6·2 per cent., which again shows a minor antigenic difference between the new type Phase 1 and *Salm. paratyphi A* H. *Salm. gaminara* has the second phase 1, 7 together with a " plus " factor and *Salm. bredeney* has the second phase 1, 7 but without the " plus " factor described by Hormaeche *et al.* (1944). *Salm. gaminara* H 1, 7 " plus " serum agglutinated the new strain and *Salm. bredeney* H 1, 7 to titre. Absorption of this serum with the new strain H 1, 7 removed all agglutinins to Phase 2 of the new strain and to Phase 2 of *Salm. bredeney* but a residual titre of 20·5 per cent. remained for *Salm. gaminara* H 1, 7 " plus ". That this residual titre was due to " plus " antibodies and not to an undescribed antibody present in the serum was proved in the investigation of another new salmonella type, *Salm. lindi* (in press). This organism has been shown to have a second phase 1, 5 " plus ". *Salm. gaminara* H 1, 7 " plus " serum from which agglutinins for 1, 7 had been removed agglutinated both *Salm. gaminara* 1, 7 " plus " and *Salm. lindi* 1, 5 " plus " to the same titre, but failed to agglutinate *Salm. bredeney* 1, 7. The serum made with the new strain agglutinated *Salm. gaminara* Phase 2 and *Salm. bredeney* Phase 2 to titre. Absorption of this serum with *Salm. gaminara* Phase 2 removed all Phase 2 agglutinins to the new type and to *Salm. bredeney* Phase 2. The H phases are represented by the symbols a — 1, 7.

This same type has now been isolated from the gut of two snakes in the London Zoo, *Eunectes murinus* (an anaconda) and *Cyclagras gigas*. In neither instance was it thought that this salmonella was responsible for the enteritis causing the death of these reptiles.

Summary

A new salmonella type has been isolated from a guinea-pig suffering from enteritis. This organism has the antigenic structure III, X : a — 1, 7 but does not contain the " plus " factor described by Hormaeche *et al.* The proposed name is *Salm. oxford*. A culture has been deposited with the National Collection of Type Cultures and is No. 8461.

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ADVICE AS TO CARE AND AFTER-CARE

J. Greenwood Wilson, M.D., F.R.C.P., D.P.H.,
Medical Officer of Health, Cardiff C.B.

" A local health authority may . . . make arrangements for the purpose of the prevention of illness, the care of persons suffering from illness, or the after-care of such persons . . ." (N.H.S.A. 1946, Sec. 28).

Circular 118/47 also said that " the local health authority " (having been *directed* by the Minister of Health to make Sec. 28 arrangements in respect of tuberculosis), " should also consider . . . firstly the desirability of making arrangements under Sec. 28 with regard to persons suffering from any other kind of illness, and, secondly, the organisation necessary for the purpose . . . '.

To make arrangements to do anything, one of the first steps is to decide who is to do it. Sec. 24 of N.H.S.A. 1946 for the first time recognises the multi-purpose health visitor who must be appointed by local health authorities, *inter alia,* . . . " for the purpose of giving advice as to the care of . . . persons suffering from illness . . .".

It has long been the policy of the Cardiff Health Authority to evolve the multi-purpose health visitor. The first step (a slow process, finally completed in 1944), was the amalgamation of the school nurse with the health visitor. The second was not to replace, when they retired one by one, the three specialist tuberculosis health visitors. True, the advent in Memorandum 266/T of a complicated scheme of money allowances for tuberculous persons necessitated a revival of specialism in this particular field for a time, but when these *ad hoc* allowances were superseded by the new social security legislation, we reverted to the system of spreading the duty of advice to the tuberculous in their homes amongst all our district health visitors, whilst still retaining the officer appointed under Memorandum 266/T as a liaison officer between the National Health Service Chest Clinic and the district health visitors. (Latterly, we have also seconded a general duty health visitor for half-time duty in our B.C.G. scheme.)

The third step, which fitted in conveniently with administrative changes in the mental deficiency section of the public health department, was to encourage the district health visitors all to take a greater interest in mental deficiency problems of care and after-care in the home though here too it was necessary to retain one specialist (but still a health visitor) to deal with difficult cases and to assist the petitioning officer in his work.

In 1944, a chat with municipal (Llandough) hospital administrators and clinicians led to a broadening of the conception of limiting home care advice to the traditional problems of the old public health and school medical services and it was decided to appoint a health visitor to carry out the follow-up work of diabetic patients discharged from hospital. Although the ideal of multi-purpose health visiting was not forsaken the complicated nature of the diabetic life made it necessary to appoint a specialist health visitor for the new work.

It was about this time too, and for similar reasons, that we also departed from principle by appointing a specialist health visitor for home care of premature infants. She received special residential training at " Sorrento ", Birmingham, but we subsequently salved our consciences for this specialist appointment by arranging special training in the care of the premature infant for all our domiciliary midwives and district health visitors at the Cardiff Premature Infants Unit (St. David's Hospital).

The health visitor selected for the diabetic work was first thoroughly trained in the after-care of these patients. She attended lectures given to medical students on diabetes and was further instructed on the daily medical round.

She visited the laboratories where glucose tolerance tests were carried out and the kitchen where diets were weighed and balanced. She read case notes and text-books until she became thoroughly conversant with the diabetic regime.

She began to visit the patients in their own homes and to understand their difficulties. She found that they lacked confidence, had fears and uncertainties about their future mode of life and that the prospect of their being disabled was a source of great anxiety to them.

Some were not intelligent enough to follow up the instructions on leaving hospital ; others could not adjust themselves to the restrictions imposed upon them by their complaint. Most of all, they suffered from lack of supervision, to ensure that they had frequent urine testing, help with dietary problems and the technique of insulin self-administration.

From these observations in the homes it became apparent that domiciliary visiting was not enough and that a diabetic after-care clinic was also necessary. It was decided to set one up at a general public health centre (maternity and child welfare and school health clinic) where a suite of rooms with necessary equipment has been allotted for this work. The rooms include one with a chiropodist's chair and equipment where a chiropodist attends at each session of the diabetic clinic.

Since the work was started in 1944, the scheme has evolved so that there are now two health visitors (one whole time and one half time), engaged on diabetic after-care working under a general physician with the help of a registrar or trainee specialist.

Diabetic patients throughout the city are covered by the scheme. Weekly visits are made to the hospitals by the health visitors who discuss the patient's problems with physicians, medical registrars and ward sisters, and learn of any special instructions that may be needed for the patient's medical after-care.

On the patient's discharge from hospital, a copy of the routine letter normally sent to his general practitioner is also sent to the health visitor at the clinic, who then visits the patient in his own home as soon as possible.

It has been found that although printed instructions are given on all essential matters such as the storage of syringes, general hygiene and care of the feet, it is more satisfactory to call at the patient's home at first in order to supervise his methods generally, and give general advice.

Many patients find difficulty in measuring their correct dosage of insulin and they get confused in the various markings on the syringe. All this is explained and the patient is reminded of the tuition he has had in hospital of injection technique, how to recognise early signs of hypoglycaemia, and the necessary precautions to be taken on occasions involving extra activity, such as the playing of games and extra house-work.

Often the patient is unable to absorb this at once and has to acquire it gradually and learn to accept this knowledge ; therefore home visiting plays a very important part in the education of the diabetic patient.

It is followed up by attendances at the clinic where the patient's way of life is checked by the physician in attendance there. Clinics are held twice a week, and it has been found that few patients have failed to attend regularly, after discharge from hospital.

A room is set apart at the clinic for instruction in urine testing. Each patient does his own testing under supervision, unless he has visual or physical defects which prevent his doing so.

As well as the regular visit of the chiropodist, the hospital dietitian also attends with the physician to follow up his advice with individual patients on details of the diet advised. In addition to the detailed advice to individual patients by the dietitian, general talks and demonstrations about catering and dietetics are given to the patients in the waiting room, appropriately illustrated by posters, leaflets and demonstration material.

In the waiting room there is also a table on which are set out display sets to illustrate the technique of insulin administration, care of syringes, weighing and preparation of diabetic dishes and the like.

Although the after-care of diabetes is perhaps the most complicated of any, the same principles have been applied in a scheme that has been gradually built up to apply to other diseases also.

The scheme was first extended to gastric or peptic cases and now also embraces paediatrics (by co-operation with the University Department of Child Health), venereal disease and asthma.

For only one disease, the diabetic have we the arrangement for the supervising consultant to attend a public health clinic. This is largely a matter of history and chance. For the other diseases, with the hospitals (and even chest clinics), being now outside local authority administration, it is administratively less complicated to send the "liaison" health visitors to work in the hospital wards and out-patient departments.

The latest development of our scheme is that with the co-operation of the mental health consultants we have chosen from our staff of general duty health visitors one with an aptitude for mental health work for a year's special training in psychiatry. At the end of her training she will become liaison officer for mental health between the district health visitors and the mental hospital with its psychiatric out-patient department at the Cardiff Royal Infirmary, exactly like the other liaison health visitors, i.e., for diabetes, peptic cases, pediatrics, premature infants, tuberculosis, venereal disease, asthma and mental deficiency. We have also offered the Cardiff Hospital Management Committee help in the follow-up of cases from the geriatric unit they propose to establish.

I anticipate the cricitism that we have departed far from the ideal with which we set out, i.e., the "multipurpose" health visitor. My answer to this is, (1) it would be physically impossible for all our 50 health visitors to troop in and out of hospitals in order to acquire the necessary follow-up angle on their cases, (2) we do maintain our original ideal by employing our *corps* of health visitors as a whole, on multi-purpose advice, (3) we shall try to adopt as far as practicable the idea suggested by Professor A. G. Watkins of the Cardiff Child Health Unit in his able appreciation of our work for that Unit ("B.M.J.", 7th July, 1951) that we should put all our district health visitors on rota for the liaison duties so that all in time get a chance of that valuable experience which is in effect a form of post-graduate training or refresher course.

I anticipate also the criticism that our health visitors may be coming between the general practitioner and his patient, but I can only say that we have not experienced any opposition or criticism from general practitioners in Cardiff. On the contrary, the Cardiff scheme has given a new opportunity for general practitioners to know and understand more about the work of health visitors. It had been hoped that they would do this through working alongside them at the health centres proposed under the National Health Service Act, but without waiting for that ideal, the achievement of which now seems to be so remote, we in Cardiff have paved the way by providing the opportunity for family doctors to discuss their cases with the health visitors. Incidentally, this may save the family doctor a great deal of time and trouble in a number of ways. Although he will have had his routine letter from the hospital on a patient's discharge, there may well be some detailed information which the liaison health visitor has learned from her visits to the hospital and which the family doctor would like to know. For him to obtain information in this way is a means of saving much time and telephoning. In the other direction, if he is being asked by, e.g., a diabetic patient for detailed advice which, in his busy life he may not have the time to give, he can refer that patient to the follow-up clinic or to the health visitor and this, in fact, is a practice which many are following. Whatever may be the position in the distant future, the family doctor has manifestly not the time under the present organisation of medical practice to give all the health education in the home which is now being given by health visitors, and as things stand there surely can be no objection to the health visitor doing this work for him.

There is another way in which by the Cardiff scheme general practitioners are learning to know and understand better the work of health visitors, and that is because the general practitioner of the future, the medical student, is working alongside the liaison health visitor as together they do the ward round with the visiting hospital consultant. This is an important long-term view.

Although not strictly relevant to the subject of this article on " care and after-care " in the sense of the follow-up of hospital patients, I should like to refer to one other experiment which illustrates further how convinced we are in Cardiff as to the almost infinite variety of service that can be carried out by health visitors. It is a conviction based on experience which makes us unalterably opposed to the suggestion that the present day basic training of health visitors requires any fundamental changes.

In Cardiff, the " Designated Officer " for " children neglected or ill-treated in their own homes " (Circulars 78/50 Ministry of Health, 157/50 Home Office, 225/50 Ministry of Education, 31.7.50) is the Medical Officer of Health. As a pilot scheme a general duty health visitor has been seconded whole time to help me in this work. She has already proved highly successful in dealing with problem families on her own, but the plan is that in due course we shall apply the " liaison " principle to her in the same way as we have for hospital follow-up and she will become the " liaison " health visitor between the health department and all the various organisations both official and voluntary that in one way or another are concerned with problem families.

In order to try and leave a clear impression of the Cardiff multi-purpose health visitor service, a table is appended showing the organisation of the health visitor staff. The table should be considered against the background that the latest census figures for Cardiff give a total population of 243,627, which according to the Registrar-General's 1950 estimate works out into age groups of 21,710 for 0-5 and 32,820 for 5-14. The number of births (live and still) in Cardiff in 1950 was 4,521 and 12 respectively.

Each district or general duty health visitor covers an approximate population of 6,400 which includes approximately 500 pre-school children and 900 school children. The health visitor staff is as follows :—

Establishment and Allocation of Duties of Health Visitors

	Establishment	
1. *Administrative*		
(*a*) Superintendent	1	
(*b*) Deputy Superintendent	½	
	—	1½
2. *Specialised Duties*		
(*a*) Immunisation against diphtheria	1	
(*b*) Open-air school	½	
(*c*) T.B. Liaison Officer	1	
(*d*) Diabetic and gastric follow-up (or " liaison ") ...	1½	
(*e*) Paediatric follow-up	1	
(*f*) Premature infant follow-up	1	
(*g*) M.D. Visitor	1	
(*h*) V.D. Clinic (Special Clinic for mothers and young children)	½	
(*i*) V.D. Follow-up	½	
(*j*) Asthma Follow-up	1	
(*k*) Porthcawl School Camp relief	1	
(*l*) B.C.G. Visiting	½	
(*m*) Child Neglect and Ill-treatment	1	
(*n*) Mental Health Follow-up	1	
	—	12½
3. *Combined Duties in Areas*		
(*a*) School Nursing	10	
(*b*) General Health Visiting	20½	
(*c*) Nursery Supervision	2	
(*d*) T.B. Visiting	2	
(*e*) M.D. Visiting	2	
(*f*) Gastric Visiting	1	
(*g*) Immunisation against diphtheria	½	
	—	38
		52

The table above expresses the work of individuals in terms of staff *time*, e.g., the V.D. follow-up health visitor (½ time), is a senior health visitor in charge of an area (see below), and also engages in general duty health visiting herself, and the pediatric follow-up (expressed in the table as " 1 " health visitor) is carried out by *two* individuals each giving half time to general duties and half time to pediatrics. The V.D. follow-up health visitor is a different individual from the one who helps at the Special V.D. Clinic for mothers and young children.

The work of the general duty district health visitors in Cardiff is arranged by dividing the city into six areas each of which is staffed by a team of a varying number of health visitors (the number varying according to volume and aggregation of the population as well as " toughness ") in each area. Each area team works with and under a senior health visitor and is based on an area clinic. To the senior health visitor is delegated by the superintendent health visitor the responsibility of running the area clinic and seeing that the work of the team which is organised at headquarters, runs smoothly, e.g., in case of sudden illness of one of its members. As far as possible the delegated area organisation embraces the work of the specialist, " liaison " health visitor, but there are exceptions in the case of tuberculosis, premature infants, mental deficiency, " children neglected, etc.", immunisation and mental health. These officers still work from headquarters. The assistance received from general duty health visitors by the whole-time specialist " immunising " health visitor is expressed in the table above in terms of the half time of one individual health visitor.

OBSERVATIONS ON THE NUTRITIVE AND THERAPEUTIC VALUES OF FRUIT AND FRUIT JUICES

H. E. Magee, M.B., D.Sc., M.R.C.P.

During the past 30 years or so big advances have been made in the manufacture of fruit juices and there are now on the market pasteurised natural juices and syrups of most of the fruits grown in this country which retain the original flavours. The juices are sold in bottles or in larger containers suitable for hotels, institutions and hospitals. The great bulk of the juices now obtainable are " straight " (i.e. unconcentrated) juices, derived from a single species or even from a single variety of fruit. For instance, juices differing widely in taste and flavour have been prepared from different kinds of apples, and the juices of different varieties have been blended to bring out particular characteristics. Similarly, juices from different species of fruit have been blended, e.g. the apple and raspberry, producing variety in flavour and colour. The scope for variation along these lines is wide. Concentrated juices have also been prepared, convenient in many ways but with the disadvantage that some of the natural flavour is lost in concentration. Syrups are especially convenient for making up drinks of varying dilutions and because of their high sugar content they will keep at room temperatures for several days after opening. Straight or concentrated juices without added sugar will spoil after exposure to the air unless kept in a cold place.

There is surprisingly little direct information about the nutritive value of fruit and fruit juices except on their vitamin contents. In some Continental countries, however, fruit purées and juices and fruit diets have long been credited with beneficial effects in a variety of diseases. It therefore seems opportune to assess the available information and to try to place fruit and fruit juices in their proper place in a nutritional sense.

In recent years fruit juices have been consumed to an increasing extent in many countries and in certain parts of Europe, especially Switzerland and Germany, consumption before the war had reached phenomenal heights. For instance, in Switzerland production of apple juice was 3.5 m. gallons in 1939 and 6.9 m. gallons in 1950. In Germany in 1939 consumption reached the huge total of 27 m. gallons. Most of the juices drunk in these countries were those of native fruits. In this country, apart from citrus juices, consumption has been limited mainly to relatively small quantities of soft fruit juices used to flavour milk shakes and ice-creams. In Switzerland and Germany apple juice predominates. These native fruit juices are free from alcohol and as a rule they are pasteurised or otherwise heat-treated, so that they will keep indefinitely unless exposed to air and warmth.

Composition. Fruit has been eaten by mankind from time immemorial, a fact significant of its health-giving value. The notion generally held about fruits was that with few exceptions they did not possess outstanding nutritive properties, but rather that, when combined with foods of higher nutritive value, they formed a pleasant and tasty item in the diet. Fruit has also been valued because it stimulates the movements of the gastro-intestinal tract. The discovery of the vitamins, the advances made in knowledge of metabolism and the significance of the mineral elements and of the acid-base equilibrium of the blood have all tended to give fruit a new importance nutritionally. Chemical analyses of fruits have been accumulating and, in addition to their vitamin contents, they have been shown to be relatively rich in basic elements bound to organic acids such as citric and acetic. Many of these organic acids are oxidised in the body to carbon dioxide and water, thus leaving the basic elements to add to the alkali reserves of the body. To this property the protective value of fruit is partly due.

Most fruits are rich in potassium, and contain appreciable amounts of sodium, calcium, iron, magnesium, and of the so-called trace elements—iodine, fluorine, copper, manganese, etc. Some, such as dates, blackberries, currants and raspberries, are particularly rich in calcium, and some, such as figs, dates, loganberries and peaches in magnesium. Deficiency states due to lack of any of the mineral elements which occur in appreciable amounts in fruits except calcium and iron, are practically unknown, and this fact has doubtless taken away from the importance ascribed to these foods. The relationship recently shown to exist between the functions of the adrenal cortex in regard to the metabolism of sodium and potassium, and the beneficial effects of sodium chloride in Addison's disease, have served to demonstrate the physiological significance of these elements.

The fresh fruits normally consumed in this country yield from about 5 to 15 calories per oz. or 80 to 240 calories per lb. ; their protein content is negligible. It is for their vitamin content, especially vitamin C and carotene, that they are chiefly prized. All contain vitamin C, but the content is extremely variable, from about 1 mg. per oz. for certain types of apple to about 57 mgs. for black currants ; the black currant is the richest of all our native fruits in vitamin C, and has no equal in this respect amongst imported fruits eaten in this country. Most of our native fruits also contain significant amounts of the B complex. The following table shows the composition of some common fruits and the corresponding juices.

COMPOSITION OF SOME FRUITS AND JUICES

Fruit	Calories per oz.	Na mg/oz.	K mg/oz.	Ca. mg/oz.	Mg. mg/oz.	Vitamins		
						Carotene IU/oz.	B1 mg/oz.	C mg/oz.
Apple	11	0·6	35	1·0	1·0	17	0·01	1–4
,, juice	14	2	25	2			0·003	2–5
Blackberry ...	8	1·1	59	18·0	8·4	90	0·01	Tr.
,, juice ...	6	—	—	—	—			Tr.
Blackcurrant ...	18	0·8	106	17·2	4·9	9–90	0·01	57
,, juice ...	12	0·9	70	8·4	3·4			28–85
Gooseberry ...	10	0·3	48	5·2	2·4	6–60	0·01	7
,, juice ...	8							6
Orange	10	0·8	56	11·7	3·7		0·02	16
,, juice ...	15	0·5	51	5·0	3·0		0·03	8–18
Victoria Plum ...	11	0·5	53	3·1	2·0	30–120	0·01	1
,, ,, juice	15							3
Raspberry	7	0·7	64	11·6	6·2	14–80	0·01	7
,, juice ...	8	0·6	44	4·0	3·4			7
Strawberry	7	0·4	46	6·3	3·3	20–200	0·01	14
,, juice ...	4	0·9	44	4·5	2·5			14

The content of nutrients in the juice is frequently higher than in the whole fruit. For instance, 100 grammes of apples yield 34 calories and 4 mgs. of calcium compared with 50 calories and 6 mgs. of calcium for the same weight of juice. On the other hand, pasteurisation reduces the vitamin C content significantly.

Physiological and Therapeutic Evidence: I know of no animal experiments to ascertain the all-round nutritive value of fruits or fruit juices, or their value in regard to particular nutrients other than vitamins. Investigations on these lines would be well worth while. In this country the notion that fruit juices are beneficial nutritionally has never been put forward with conviction and authority; it is held only vaguely and only in respect of vitamins have they been commended. For this reason fruits like apples, which are relatively poor in vitamins, have been rated very low in a nutritional sense. Indeed, during the late war when there was some uncertainty about the sufficiency of vitamin C in the national diet, views were often expressed, which we must now regard as somewhat extravagant, about the low food value of the apple in particular. It was termed a " filler " the only advantages of which were a pleasant taste and a laxative action. Nevertheless people continued to remember the old adage—" An apple a day keeps the doctor away ". Apart from the rare cases of scurvy we cannot point to any clinical evidence worth consideration obtained in this country which throws any light on the value of fruits in a therapeutic sense, except perhaps the banana, for which much success has been claimed in coeliac disease. On the other hand on the Continent there is a belief, extending back to the days of Tissot in the 18th century, backed by much recorded experience, that many fruits possess considerable therapeutic properties.

Hyperpyesia: Heupke of Frankfurt a/M. has published a semi-popular book (1949) on the use of fruit and fruit juices in the treatment of diseases of the blood vessels, heart and kidneys, alimentary system, and of gout. He lays down detailed directions for the preparation of diets containing fruit and fruit juices, and the duration of the treatment. He recommends that the course of treatment should last not more than about a week at any one time, or alternatively, that fruit days (Obsttage) should be given at weekly or longer intervals over seven weeks. On fruit days 2-3 lbs. of fresh fruit are given, or up to 1 litre of juice, providing 600-700 calories and up to about 10 grammes of protein. Fruit days of this sort are really fast days and they obviously could not be kept up long with impunity. To increase the energy content of the diet he adds nuts and dried fruit in suitable amounts. The distinctive feature of the fruit diet is its high content of potassium relative to sodium. In our ordinary mixed diets the potassium-sodium ratio is of the order of 1:2 compared with 6:1 for the fresh fruit diet, and 13:1 for the fruit juice diet. The fruit diet is, of course, very poor in acidic elements, phosphorus, sulphur and chlorine. According to Heupke the change in the potassium-sodium ratio plays an important part in the treatment of renal-circulatory syndromes and in hyperpyesia. The beneficial effect he attributes to the diuretic effect of potassium which causes withdrawal of sodium chloride from the body and in turn diminishes the volume of blood and the amount of water in the tissues. Potassium citrate has been recognised as a diuretic and potassium acetate as a diaphoretic for many years and it seems reasonable to suppose that these salts in fruit juices would act in the same way as they do when given in pure form therapeutically. Richards and others (1927) showed that the addition of potassium citrate to the diet of young pigs diminishes absorption and retention of nitrogen, calcium and phosphorus, and that sodium had the opposite effect. They found no evidence to substantiate the view formerly put forward by Bunge that excess potassium drained sodium and chlorine from the body. On the other hand Chaney and Blunt (1926) found that additions of citrus fruit to the diet of children increased the retention of calcium, phosphorus, magnesium and nitrogen, and also the rate of growth. Eddy (1933) found that bananas also increase the retention of calcium in boys and in rats, and Lanford (1942) showed that potassium citrate and citric acid in amounts similar to those found in lemon juice stimulated, though to a less extent, retention of calcium.

Several authors have reported success in the treatment of diseases of the heart and blood vessels with fruit or fruit juice. Bock and Lebsanft (1942) compared the effects of fruit juice, salt-free and ordinary hospital diets on patients with essential hyper-tension. In 97 patients on the fruit juice diet the systolic blood pressure fell on the average by 67 mm. and the diastolic by 36 mm., compared with 44 and 25 for 105 patients on the salt-free diet, and 34 and 20 mm. respectively for 69 patients who were not treated dietetically. Gutzeit (1928) found fruit and vegetable juices of great benefit in cases of failing compensation and angina. Hahn (1943) found more improvement in the electro-cardiograms in patients treated on fruit juice diets than in those treated on salt-free diets. Dick and Schwarz (1947) showed in dogs that the Kempner rice-fruit diet (250-350 gm. rice 750-1,000 cc. fruit juice or fruit and 100 gm. sucrose) caused significant decreases in the blood pressure of dogs affected for two years with experimental hyper-tension. Blöch (1950) has reviewed recent published work on the Kempner diet in hyperpyesia. Of 71 cases of essential hyper-tension treated, 67 per cent. were improved and 33 per cent. unchanged ; both the systolic and diastolic fell in the former after a very variable period—a few days to several months, and in some cases the heart showed remarkable decreases in size. More recently this mode of treatment has been tested clinically by a committee specially appointed by the M.R.C. (1950). The diet consists only of rice, fruit (1,500 gm.) and sugar with no added salt or fat, and yields some 2,000 calories and 20 grammes of protein daily. It was given to 44 cases of benign, malignant and renal hyper-tension. Significant lowering of the blood pressure occurred after a few days but rose again when the diets changed. Sodium seemed to be the most important pressor factor in the diet because when the sodium chloride was increased even on the rice-fruit diet, the blood pressure rose rapidly. One of the renal cases developed uraemia and died while on the diet ; it is therefore not advised for the treatment of renal hyper-tension. All the patients lost weight ; none of them liked the diet and about six weeks was as long as any of them could tolerate it.

Peptic Ulcer. Heupke claims much success in the treatment of peptic ulcers with fruit and fruit juices. He recommends one litre of juice of apple, black currant, strawberry, blackberry, cherry or grape for about five days, after which the patient is put on an ordinary peptic ulcer diet. Grape juice requires dilution to avoid harmful osmotic effects ; it contains some 18 per cent. of sugar.

Tests of the effects of fruit juices on secretion of the gastric juice have given variable results. Of 12 papers consulted in which the effects of fruit juice on cases of peptic ulceration are recorded, three report increases in acidity, three no change, and two decreases ; one that fruit juice delayed the emptying time of the stomach ; one that it improved digestion of milk, two that it caused some relief of pain and one where there was no change in symptoms.

Diarrhoea. Treatment of acute diarrhoea by means of the " apple cure " has, according to Heupke, had very successful results in recent years. This treatment was introduced by Tissot in the 18th century and it was revived in more recent times by von Moro and Heissler. The beneficial effect of the apple purée is attributed mainly to its pectin content and the acid reaction. The pectin swells and engulfs the bacteria causing the diarrhoea and the acid reaction prevents their further growth. Dried blaeberries given orally have been used as a household remedy for diarrhoea for many years; the fresh berries and the juice are also beneficial, but the dried berries are claimed to be more effective. Chronic diarrhoea did not respond with the same degree of certainty to fruit treatment; the banana was found to be the most effective.

Steatorrhöea. In sprue good results have been obtained with strawberries, especially when fruit days are repeated at regular intervals over several weeks. Bananas, fresh or dried, have long been recognised in this country as of great value in steatorrhöea. Heupke recommends a combination of fresh fruit juice and grated apple for sprue.

Fever. In fevers of short duration a day or two of fasting is often of great benefit; the body is enabled to get rid of waste products and there is little desire for solid food of high energy-yielding type. But there is generally a thirst that is difficult to satisfy and for this fruit juices seem to be the most suitable. By their excess of base they neutralise acid metabolites and by supplying relatively large amounts of mineral salts and vitamins they exert their "protective" influence on the body as a whole. Many fruit juices are far from negligible also as sources of energy; for instance, two pints of apple juice provide about 600 calories. The fruit juices are acceptable to most patients.

In fevers of long duration rigid fruit days are contra-indicated, because of the need to maintain the patient in the best state of nutrition possible. Fruit juices, however, still have their place for the relief of thirst, and because of their protective value and acceptability; for such fever patients a variety of fruit juices is desirable.

Summary

1. The literature on fruit and fruit juices has been examined from the point of view of their value in health and disease.

2. In recent years there has been a tendency to evaluate fruit and fruit juices exclusively in relation to their vitamin content. Nutritive estimates have consequently been made of many fruits, particularly certain native ones, which are not in accordance with what is known of their composition and their physiological and therapeutic effects.

3. The physiological evidence goes to show that fruit or fruit juices add to the variety of diet, improve palatability and stimulate the peristalsis of the alimentary canal; that they help to maintain the alkali reserve of the blood, to neutralise acid metabolites and make a significant contribution, especially fruit juices, to the energy intake. The high potassium : sodium ratio also plays a part in keeping the volume of the blood within normal limits.

4. There is evidence, dating back some 200 years, that apples and blaeberries are of value in treating the acute forms of diarrhoea. Strawberries have been found to have good effects in sprue, bananas and fruit juices in steatorrhöea, and a variety of fruits and juices in peptic ulcer. Considerable success has been claimed in the treatment, with a variety of fruits and fruit juices, of hyper-tension of the essential type or arising from cardiac and renal conditions. Experience with the Kempner rice-fruit diet seems in this country to have given only temporary relief, whereas on the Continent more

permanent results have been claimed. In febrile conditions of any sort fruit juices form an acceptable and refreshing drink which can add appreciably to the energy intake and at the same time exert a generally protective effect.

5. Most of the physiological and clinical evidence of fruit and fruit juices has come from Continental countries; it is highly desirable that the claims made should be tested in this country.

6. The consumption of native fruit juices on the Continent has attained very high levels in recent years. Big advances have been made in the manufacture of fruit juices in this country and many varieties and blends of fruit juices can be obtained in various types of container in natural or concentrated form or as syrups. Most of these juices are pasteurised or sterilised and most of those in natural form retain all the flavour of the original fruit.

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TREATMENT OF ANTHRAX, BOTULISM AND SNAKE-BITE

A number of alterations and additions have been made to the list originally published in this Bulletin in January, 1950, of centres which keep emergency supplies of sera for the treatment of anthrax, botulism and snake-bite. A revised list is therefore printed below. A 24-hour service is maintained at each centre.

Centre and Telephone Number

The Dispensary, Newcastle General Hospital	Newcastle 35211
The Dispensary, Cumberland Infirmary, Carlisle	Carlisle 590
Seacroft Hospital, York Road, Leeds	Leeds 45194
Castle Hill Hospital, Cottingham, York	Cottingham 47372
Nottingham City Hospital, Hucknall Road, Nottingham.	Nottingham 66292
Regional Blood Supply Depot, Brookland Avenue, Cambridge—	
Monday to Friday 8 a.m.—10 p.m.	Cambridge 2536
Saturday 8 a.m.— 6 p.m.	
All other times	Cambridge 87988
North Middlesex Hospital	Tottenham 3071
South London Blood Supply Depot, Stanley Road, Sutton, Surrey.	Vigilant 0068
The Dispensary, Royal Southants and Southampton Hospital (Snake anti-serum only).	Southampton 76211
Northampton General Hospital	Northampton 4680
Royal Berkshire Hospital, Reading	Reading 2231
Ham Green Hospital, Bristol	Bristol 31165
Swilly Isolation Hospital, Plymouth	Plymouth 4311
Pathological Department, Royal Devon and Exeter Hospital.	Exeter 3519
Pathological Department, Royal Cornwall Infirmary, Truro.	Truro 3029
Canton Infectious Diseases Hospital, Cardiff	Cardiff 960
The Dispensary, Selly Oak Hospital, Birmingham	Selly Oak 1361
The Dispensary, Manchester Royal Infirmary	Ardwick 3300
The Fazakerley Isolation Hospital, Liverpool	Aintree 2324

In addition, 10-ml. containers of anthrax anti-serum for use *prophylactically* in persons exposed to infection may be obtained during the day-time from the following centres:

Public Health Laboratory, 16-18, Edmund Street, Bradford.	Bradford 24314
Public Health Laboratory, 184, High Street, Kingston-upon-Hull.	Hull 35371
Bacteriological Department City Laboratories, 126, Mount Pleasant, Liverpool.	Royal 3636
Central Public Health Laboratory, Colindale Avenue, London, N.W.9.	Colindale 6041 and 4081
Public Health Laboratory General Hospital, Westgate Road, Newcastle 4.	Newcastle 34920
Public Health Laboratory, General Hospital, Northampton.	Northampton 347
Public Health Laboratory, County Hall, Taunton	Taunton 3451
Public Health Laboratory, Institute of Preventive Medicine, The Parade, Cardiff.	Cardiff 8288

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

AUGUST, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

	August 4th	August 11th	August 18th	August 25th	Average weekly figures for August, 1950
Scarlet Fever	646	468	479	463	725
Whooping Cough	2,905	2,661	3,165	2,948	3,693
Diphtheria	33	19	31	23	42
Measles, excluding Rubella ...	4,944	3,621	3,252	2,352	6,053
Acute Pneumonia	312	223	233	181	235
Meningococcal Infection ...	29	24	25	31	22
Acute Poliomyelitis (Paralytic)...	55	55	57	60	320
,, ,, (Non-paralytic)	62	58	63	56	155
Ophthalmia Neonatorum ...	31	25	34	40	38
Puerperal Pyrexia and Puerperal Sepsis	167	190	249	240	38
Dysentery	193	149	153	174	167
Paratyphoid	34	44	52	46	6
Typhoid	4	12	10	6	8
Smallpox	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street,
Westminster, S.W.1.

EPIDEMIOLOGICAL INVESTIGATION AND RESEARCH:

THE RELATIONSHIP BETWEEN THE DISTRICT MEDICAL OFFICER OF HEALTH AND THE PUBLIC HEALTH LABORATORY SERVICE*

G. S. Wilson, M.D., F.R.C.P., D.P.H.
Director of the Public Health Laboratory Service

To-night I want to clear away one or two misconceptions that have arisen about the relationship of the Public Health Laboratory Service to the Medical Officer of Health, and to suggest ways in which co-operative research investigations might profitably be carried out between them.

The National Health Service Act and the Medical Officer of Health

It is often stated that the National Health Service Act has dealt a severe blow to the cause of preventive medicine. I am not one to take as gloomy a view as that, nor do I wish to enter into the arguments for and against this proposition. But I do wish to consider for a moment, as objectively as possible, the bearing the Act has on the medical officer of health. Removal of the control of infectious diseases hospitals from his care is in itself perhaps no great loss, since the medical officer of health should not be primarily interested in the therapeutic treatment of sick persons. This deprivation of function, however, has made it clear that the medical officer of health is not to be regarded as a clinician, and cannot therefore be looked to by the general practitioner for help in the diagnosis of infectious disease. The rift caused in this way between the medical officer of health and the general practitioner has been widened still further by the right which the general practitioner now enjoys under the Act of being able to call into consultation clinical specialists attached to hospitals in the Region. The effect of this will undoubtedly be to bring the general practitioner into closer contact with the hospitals, and incidentally to increase his interest in the diagnosis and treatment of disease. The bond between the general practitioner and the hospital consultant will get stronger, to their mutual benefit, and the clinical relations that used to exist between the general practitioner and the medical officer of health will gradually disappear.

Though the medical officer of health is no longer responsible for the care of the individual patient, he is still responsible for safeguarding the health of the community. He may no longer be in charge of sick beds, but he is left with the opportunity of investigating disease and of studying the broad problems of health and environment.

The Public Health Laboratory Service

Under the Act a new service has been created—the Public Health Laboratory Service—whose duty it is, in the words of Section 17 of Part II of the Act, to take part in " the control of the spread of infectious diseases ". Both the medical officer of health, therefore, and the new laboratory service are essentially concerned in the prevention and spread of infectious diseases ; and the thesis that I want to put forward is that if Preventive Medicine, in which at one time Great Britain led the world, is to regain its former place, the medical officers of health and the Public Health Laboratory Service must join hands as allies and friends in co-operative research into the epidemiology and control of infectious disease.

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* A paper read before the County District Group of the Society of Medical Officers of Health at the Annual Meeting at Southport, 1951.

Contributions of Bacteriologists to Epidemiology

At this point I may hear murmurings of dissent. I may be accused of gross presumption in suggesting that a Service which, even with its emergency progenitor, is scarcely more than ten years old, should claim alliance and even friendship with a distinguished service which has recently celebrated its centenary. How am I to justify myself? I must try to do so by pointing out that, though many of the bacteriologists who are fundamentally interested in Preventive Medicine have only recently been organized into a national service, bacteriologists have in the past made considerable contributions to our knowledge of the causation and mode of spread of infectious disease. May I quote a few examples?

Let us take first of all Weil's disease. The type of infectious jaundice which this name signifies is of worldwide distribution ; it occurs mainly in sporadic form, but rises at times, as in the American war of Secession, to epidemic proportions. The causal organism was discovered by Inada and his colleagues in Japan in 1915 ; and much of the epidemiology of the disease, including its occurrence in the trenches of the first world war, in the coal-mines, in sewer workers and in fish washers, as well as the latent endemic infection among rats, was worked out by bacteriologists in company with clinicians, and is associated with such names as those of Uhlenhuth, Adrian Stokes, Buchanan, Schüffner, Alston, and John Smith. The further demonstration that human cases in this country may be caused by two different types of organism—*Leptospira icterohaemorrhagiae* and *Leptospira canicola*—the one derived from rats, the other from dogs, we owe in large measure to another laboratory worker, Dr. Broom.

Undulant fever affords a further example of a disease whose bacterial causation and mode of spread were discovered by bacteriologists. Neither of the two epidemiologists who investigated the disease in Malta at the time of the Mediterranean Fever Commission's inquiry was able to throw any light on the means by which *Brucella melitensis* gained access to the human body. Indeed, both of them reached the conclusion that milk did not appear to have any relation to its spread. It was the Maltese bacteriologist Zammit who, conducting his investigations in the field, pointed out the true vehicle of infection by his discovery of widespread dissemination of the causative organism among goats and its excretion in their milk. Following up this disease, I may remind you that the demonstration of brucella infection among cattle due to *Brucella abortus* was made by Bang and Stribolt in Denmark ; that the occurrence of human cases of undulant fever caused by *Br. abortus* was pointed out by Bevan in Southern Rhodesia ; and that the discovery of endemic disease in Great Britain was made by clinicians and bacteriologists who went into the field to collect their own material.

As a more recent example I would refer to the demonstration in this country of the existence of Q fever. As you will remember, the fundamental work on this disease, including the finding of the causative organism, *Rickettsia burneti*, and the existence of an endemic reservoir of infection in cattle, was carried out by two bacteriologists in Australia—Derrick and Burnet. When the disease appeared in Italy during the late war, it was studied by a large team of American investigators, as well as by two of our own specialists—Caughey and Dudgeon. In the summer of 1949 suspicious cases occurred at the Royal Cancer Hospital which were proved by MacCallum at the Virus Reference Laboratory at Colindale to be cases of genuine Q fever. The subsequent epidemiological investigations, with their revelation of endemic foci of infection in cattle and the presence of *R. burneti* in the milk of cows supplying the original patient, were carried out by two bacteriologists, Stoker and Marmion, working in co-operation with the local medical officers of health.

I might continue this list, but I do not want to weary you. Let me recall to your mind, however, the important part played by Colebrook and his colleagues at Queen Charlotte's Hospital in working out the exogenous mode of spread of puerperal infection; of Allison, Cruickshank, Miles, Spooner, and others in demonstrating the ways by which cross-infection spreads in hospitals—an accomplishment which was possible only by the use of the finger-printing methods that have been devised in the laboratory for distinguishing between different strains of haemolytic streptococci and of staphylococci; and of the late Stanley Griffith's great work in defining the part played by raw cow's milk in the transmission of non-pulmonary tuberculosis. I cite these examples, among others, to prove to you that substantial increases in our knowledge of the causation and mode of spread of infectious disease have been made by bacteriologists working in the laboratory and the field. In soliciting, therefore, the collaboration of the medical officers of health, the Public Health Laboratory Service does not come empty-handed. It has something to contribute to the pool. What we have to decide is how collaboration can prove most effective.

Joint Investigation of Outbreaks

The medical officer of health is responsible for safeguarding the public health of the community which he serves. It is his duty to look into the occurrence of cases or outbreaks of infectious disease, to study their origin, to suggest means by which they may be prevented, and to take such measures of control as he considers necessary or practicable. To do this thoroughly, he must have access to a good laboratory service. The basic principles in the control of infectious disease are removal of the source of infection or interference with its mode of spread. To fulfil either of these purposes it is necessary to know what the causative organism is and where it is. Only the bacteriologist can give the answer to these questions. This is not to say that intelligent action may not be taken in the light of previous knowledge without calling in bacteriological assistance, but it does mean that any medical officer of health who takes this action is depriving himself of what may prove to be vital information to the success of the measures he institutes. Who, now, would be shortsighted enough to attempt to investigate the mode of origin or spread of an outbreak of typhoid or paratyphoid fever without making full use of the Vi-phage typing methods worked out by Craigie and Felix, which have so often revealed a relationship or a difference that would otherwise have not even been suspected? When the Sheffield outbreak of paratyphoid fever in the summer of 1946 followed closely on that at Halifax, what a lot of time was saved in the epidemiological inquiry that followed by Felix's report that the two outbreaks were caused by two different Vi-phage types of organism! How often has the laboratory shown that a midwife or a matron or a nurse was the reservoir of infection in a continued outbreak of streptococcal or staphylococcal infection in a maternity home or nursery; or that an apparently healthy canteen worker was responsible for contaminating a large batch of food; or that a child admitted for some other illness was disseminating dysentery bacilli throughout a ward? I need not pursue this argument, because I know that most, if not all of you, are convinced of its truth. The question, therefore, that arises is under what terms shall the laboratory be asked to help.

Before the war, when the medical officer of health had his own laboratory or relied on a commercial laboratory situated at a distance, it was common for public health bacteriologists to be regarded as technicians whose function it was to perform certain stereotyped tests and report the result in due course. Most of that is now changed. The bacteriologist is, or should be, called in to help the medical officer of health in all epidemiological investigations into infectious disease ; and he should be called in as an equal. The old method of submitting specimens to the laboratory and expecting a report without allowing the bacteriologist to make himself familiar with the circumstances in which the specimens were collected is rather like telling a surgeon to remove the appendix without giving him the opportunity of examining the patient's abdomen. In collaborating in this way, however, the bacteriologist does not wish to interfere with the responsibility of the medical officer of health for the control of infectious disease in his area. He comes in essentially as an adviser, but as an adviser who must be given a fairly free hand to make such investigations as his expert knowledge dictates to be necessary. The trained bacteriologist has spent many years studying the ways and habits and distribution of bacteria, and he is best qualified to ferret out the location of the causative organism in most epidemic manifestations of infectious disease. In carrying out this highly specialized work he will naturally be dependent on the knowledge possessed by the medical officer of health of the supply and drainage services of his district, of the organization of schools and institutions, of the abattoirs, factories, shops, canteens, and restaurants associated with the preparation and distribution of food, of the customs and habits of the people, and of the medical officer's own past experience in the investigation of similar problems. Much of the value of the inquiry will be lost if there is not full exchange of information between the medical officer of health and himself, and only by true collaboration can the combined knowledge and experience of the Health Department's staff and the laboratory be fully utilized and the problem at issue dealt with most effectively.

Initiation of Epidemiological Research

In all this the bacteriologist comes in at the request of the medical officer of health. There are times, however, when the bacteriologist wants to initiate an inquiry on his own, and here we tread on rather dangerous ground. There is still, I regret to say, a residue of public health workers who regard epidemiology as their own prerogative. They regard any attempt by a bacteriologist to undertake epidemiological research as an encroachment on their own field. Why is this? Epidemiology is certainly one of the subjects in which public health workers are generally interested, though it is true to add that not every medical officer of health is specially drawn towards it ; there are many other ways in which he may manifest his interest in the public good. But epidemiology is a subject, like every other department of science, which should be free for investigation by all and sundry. Many of the greatest advances in our epidemiological knowledge have been made by workers who were not associated with the public health service. Edward Jenner was a field naturalist ; William Budd was a general practitioner ; John Snow was an anaesthetist ; Pasteur was a chemist ; Ronald Ross was an Army surgeon ; Savage's work on food poisoning was done in his capacity as a laboratory investigator ; and Pickle's work on infective hepatitis in his rôle of rural practitioner. Some of the epidemiological contributions made by bacteriologists have already been referred to, and I need not repeat them. No one familiar with the history of science can fail to realize the advantage of approaching a problem by numerous different ways. Medical officers of health are peculiarly well situated to provide opportunities for field investigations which they do not propose to undertake themselves, and nothing but good can come of such enlightened generosity.

Epidemiology presents numerous facets, and one of them appeals to the bacteriologist. The view sometimes expressed that the bacteriologist should be content to work in his own laboratory ignores the fact that his interest in bacteria cannot be confined to the test-tube. He wants not only to study their growth on artificial media, but to observe how they behave in the human and animal body, what reactions they give rise to in the individual patient, and how they spread when introduced into the herd. To satisfy this natural curiosity he should be permitted access to material in the field, and he should be helped as far as possible to carry out experimental investigations in which some of the variable factors that normally complicate his observations may be partly or completely controlled. I want to make a plea, therefore, that no obstacle should be put in the way of any reasonable request by the bacteriologist to carry out research work in the field. How much less should we know about the behaviour of infectious disease in this country and the tropics if the bacteriologist had been confined to the precincts of his own laboratory.

I hope that I have now made my position clear. In the investigation of an individual outbreak confined to the area over which a given medical officer of health has jurisdiction the bacteriologist is called in as an expert colleague. He should be allowed to study the background of the outbreak, to collect his own material for laboratory examination, and to undertake such other investigations as he considers necessary. Provided that he works in the closest contact with the medical officer of health and his staff and refrains from the impracticable or the inexpedient, there should be no danger of overlapping or confusion, and no infringement on the rights of the medical officer of health. When, on the other hand, the bacteriologist wishes to initiate research work in the field, then he should ask the medical officer of health, who, in his turn, should afford him all reasonable facilities. The relationship between the two was discussed by Dr. Gibson at last year's congress, and I am in full agreement with what he said.

Regional Epidemiologists

One further misunderstanding should perhaps be disposed of. The Public Health Laboratory Service has been accused of appointing its own regional epidemiologists with the intention of depriving the local medical officer of health of his proper work. It is true that three or four regional epidemiologists have been appointed, but with no such intention in mind. Assuming that all local medical officers of health and local laboratories will want to carry out their own epidemiological work, there is still ample room for a few whole-time epidemiologists attached to laboratories. Their purpose can perhaps be enumerated in some such way as the following:

(1) To help any area in the region in the investigation of an outbreak that is beyond the local resources.

(2) To investigate local outbreaks or cases of disease more intensively than would otherwise be possible. For example, a careful study might

be made of each sporadic case of salmonella infection ; the local laboratories and medical officers of health have seldom time to do this properly.

(3) To co-ordinate the investigation of outbreaks which spread over an area served by two or more laboratories.

(4) To investigate outbreaks of special importance for which a whole-time worker may be required for some time, such as the Oswestry outbreak of typhoid fever or the Sheffield or Eastbourne outbreaks of paratyphoid fever.

(5) To keep an eye on all epidemiological happenings in the region, and to follow up anything that appears to be of particular interest in co-operation with medical officers of health and local or reference laboratories.

(6) To help in organized field trials, such as those on vaccination against whooping cough or the use of gamma globulin in the prevention of measles.

(7) To take part in special epidemiological surveys, such as the present poliomyelitis survey.

(8) To help sub-committees or working parties interested in the study of particular problems concerned with infectious disease.

(9) To participate in basic epidemiological research of a type that can be dropped for a time if more pressing problems call for attention ; for instance, the distribution of *Salmonella* in poultry, animals, or other hosts, the rôle of nasal carriers of streptococci in the spread of disease, or the period of survival of pathogenic organisms under different environmental conditions.

From this list I think you will agree that there is plenty of work to occupy a few whole-time epidemiologists in the country without infringing on the duties proper to a medical officer of health. Much of this work will, of course. be done in close association with medical officers of health, but none of it, with the exception perhaps of the investigations listed under (2) and (9), could be carried out by any one medical officer of health in his own limited area.

Limitations of Research by Medical Officers of Health

Having now, I hope, proved to you conclusively that the main interest of the Public Health Laboratory Service in epidemiology is research, and that the Service has no desire whatever to interfere with the responsibilities of the medical officer of health for the administrative control of infectious disease in his area, let me now pass on to make a few constructive suggestions.

The public health service as at present constituted is handicapped in epidemiological work by the fact that the area of the local health authority is often too small a unit in which to make significant observations. This, of course, is a general criticism ; it has some exceptions. For instance, localized outbreaks of disease can be fully investigated and may at times yield illuminating results. Various methods for the control of epidemic disease in residential nurseries and similar institutions can be explored. McGonigle's work on the relation between housing and disease affords an excellent example of what can be done even in a restricted area. Broadly speaking, however, generalized conclusions on the behaviour of disease can be drawn only from the survey of a wide field. Many outbreaks cover an area extending over several health authorities and cannot be properly followed up by any one medical officer of health. More important, however, is that in the genesis of disease, particularly disease as it affects the community, so many variable factors play a part that it is almost impossible to unravel them unless they can be observed operating in different proportions. The analogy is with a differential equation in which one or more of the terms may be standardized experimentally or may be shown by observation to remain approximately constant, thus opening the way to a definition of the rest. What then is the remedy? There seem to me to be two alternatives. Either the epidemiological unit must be topographically enlarged, or the present small units must be functionally enlarged. The public health service might be organized on a regional basis and a medical officer of health appointed to a post corresponding to that of the Senior Administrative Medical Officer of the hospital service. Alternatively, medical officers of health in their own restricted areas might combine with each other in collective observations on specific problems. You are, of course, much better qualified than I am to discuss the administrative advantages and disadvantages of the two methods, but I am very doubtful myself whether the scientific gain that might result from organizing epidemiology on a regional basis would more than counterbalance the ill effect of severing relations between the medical officer of health and the other employees of the Local Authority that are interested in personal and environmental health or of losing the confidence of the public which a local medical officer of health enjoys.

Joint Epidemiological Research

However, I do not want to look too far into the future to-night. Accepting the present structure of the public health service, I suggest that a great deal of valuable epidemiological work might be carried out by the simple process of collaboration. By this means it is possible for the individually small contributions of medical officers of health to be fitted together to yield conclusions that are both general and significant. Already, co-operative inquiries have been set on foot between medical officers of health and the Public Health Laboratory Service and Medical Research Council, and already some of them have yielded fruit. Let me quote five instances: (a) *Measles Globulin trials.* These trials, an account of which was given in the Lancet of 9th December, 1950, were carried out in collaboration with medical officers of health in Middlesex and the London County Council ; in Salford, Manchester and Liverpool all the work was done by the staff of the Health Department according to a uniform plan worked out in conjunction with Professor Bradford Hill. (b) *P.T.A.P. trials.* These trials, which are not yet completed, are again being carried out by the medical officers of health and staff of twelve county boroughs in accordance with a plan carefully prepared beforehand. (c) *Whooping Cough vaccination trials.* These trials were begun in a small way by laboratory workers who made controlled observations in residential nurseries ; but, when it became clear that observations on a much larger population would be required, medical officers of health were approached in various places—Tottenham, Edmonton, Wembley, Walthamstow, Leyton, West Ham, Manchester, Leeds, Oxford, Cardiff and Poole—to find out whether they would be prepared to collaborate in a large-scale investigation to be made on children attending the infant welfare clinics. Three of the regional epidemiologists of the Public Health Laboratory Service —Dr. Cockburn, Dr. Payne, and Dr. Bolz—are taking part in this investigation, but the bulk of the work is being carried out by the staff of the public health departments themselves, strengthened when necessary by health visitors and nurses financed by the Medical Research Council. The response from the medical officers of health has been magnificent, and I believe that this will prove to be one of the most valuable epidemiological inquiries ever made in this country. (d) *Inoculation and Poliomyelitis survey.* This survey has just begun. Before inaugurating it, the Medical Research Council went straight to the Society of Medical Officers of Health, where they received most cordial promises of co-operation and most valuable constructive criticism. The President of your Society agreed to sign jointly with the Secretary of the Medical Research Council a letter addressed to medical officers of health throughout the country; and the replies which this letter has called forth leave no doubt of the willingness and latent enthusiasm of many medical officers of health to take part in a research investigation on a national scale. (e) *B.C.G. and Vole bacillus vaccination trials.* In order to try and determine what is the value of vaccination with B.C.G. or with the vole bacillus in diminishing the incidence in this country of clinical tuberculosis after primary infection, the Medical Research Council has just launched controlled trials in three areas—part of the Home Counties, the Birmingham region, and the Manchester-Leeds region. Tuberculin-negative school leavers are to be vaccinated and re-examined at intervals by radiography and tuberculin-testing for three years. The follow-up in this investigation is expected to prove very difficult, and only by the whole-hearted co-operation of the medical officer of health and his staff is success likely to be achieved.

Group Research by Medical Officers of Health

Apart from such inquiries carried out in association with the Public Health Laboratory Service, I think that a good deal of valuable work could be done by groups of medical officers of health themselves. Let me make one or two suggestions. (*a*) It is surprising how defective our knowledge still is of the exact period of infectivity of some of the common diseases, such as measles, mumps, rubella, and the common cold. No one medical officer of health can hope to make sufficient observations on any of these diseases to justify the drawing of general conclusions; but if interested medical officers of health throughout the country were to co-operate in a planned inquiry, a notable addition to our knowledge might be made. (*b*) One subject in which I have been trying to interest clinicians and epidemiologists alike is late adult male pulmonary tuberculosis. It is not always realized that, though the mortality from pulmonary tuberculosis in females begins to decline after the age of about 25 years, in males it continues to increase till it reaches its maximum about 60 years. The total mortality rate from pulmonary tuberculosis is almost twice as high in males as in females; and the disease, by incapacitating and destroying the skilled worker, constitutes a grave menace to our industrial economy. Little seems to be known about the factors responsible for the great differences between the sexes, or even about the topographical, social, or occupational incidence of the disease. There is surely here a problem worthy of attention. (*c*) Dr. Gibson pointed out in his address last year that epidemiological investigation need not be restricted to infectious disease. A careful study of social and environmental conditions might well throw light on the aetiology and prevalence of such diseases as coronary thrombosis, duodenal ulcer, or carcinoma of the lung. (*d*) One of the grave defects of our present system of registration is that an occupational census is made only once in ten years, and that the results of it are not published till some years later. I wonder whether it would not be possible for medical officers of health to make their own occupational census and to keep a running analysis of occupational mortality. How this might be done is a matter for discussion, but the knowledge, if it was obtainable, would undoubtedly prove of use not only to statisticians but to many workers interested in the distribution of disease. (*e*) The problem of dental caries in children has already prompted some public health inquiries, but I feel that a great deal more might be done in collaboration with the dental service to clarify its complex aetiology. Residential institutions are admirable places in which to make controlled observations on the effect of changes in diet, and a co-operative survey in different parts of the country might well reveal the existence of predisposing or exciting factors the importance of which has so far been overlooked or underestimated. (*f*) Much useful information, I believe, might be acquired by co-operation with the general practitioner in the epidemiological field—especially in rural areas. Local epidemiological societies have been formed in Essex, Middlesex, Devonshire and possibly elsewhere. Their success must depend on the intelligence, keenness and perseverance of the members, and too much should not be expected of them. Their possibilities, however, have not yet been fully explored, nor the technique of their working sufficiently studied to predict the extent of the contribution they might make.

Planning Research : the Experimental Approach

If working parties of medical officers of health are to be established for purposes such as those I have just outlined, it should be borne in mind, firstly that careful planning of a joint inquiry is needed, preferably with direct help from a statistician ; secondly that the inquiry must be diligently and conscientiously pursued ; and thirdly that in some types of investigation nothing can replace the first-hand qualitative observations of the trained epidemiologist. I am becoming more and more sceptical about the value of the questionnaire filled in mechanically by a multiplicity of persons varying in their interpretation of the main questions at issue. A preliminary questionnaire may be of use in helping to define the problem ; but information of real value is likely to be gained only by skilled observers devoting themselves personally to the task and conforming strictly with definitions that are agreed on beforehand. For investigations such as these a small team of workers in constant touch with each other doing first-hand research of high quality is much better than a medley of persons operating individually or doing most of their work by proxy.

I am inclined, too, to doubt whether pure observational epidemiology has much more to teach us. I do not want to discourage its practitioners in any way ; but just as in the laboratory sciences the experimental method has proved most fruitful, so I believe that in field epidemiology the introduction of an experimental approach, whenever possible, should be attempted. More rapid progress, I suspect, will be made in this way in studying, for example, the control of institutional outbreaks of Sonne dysentery by different means than by merely taking the usual stereotyped measures and observing the result. There are many human diseases that cannot be reproduced satisfactorily in laboratory animals. In my opinion controlled observations on the human population are practically essential for the solution of certain problems ; and the progressive public health department should not only develop the mechanism by which these may be made, but should by its manifest interest in the welfare of its people command sufficient confidence to enable them to be carried through successfully. In cultivating the experimental approach to the various problems that confront him the medical officer will inspire the whole of his staff and provide them with a purpose which cannot fail to be productive of results.

Conclusion

In conclusion, let me reaffirm my belief in the value of co-operation between medical officers of health and the Public Health Laboratory Service. The part played by environmental factors in the causation of epidemic disease presents a wide field of opportunity for those who care to explore it. Either medical officers of health working alone or in groups, or the Public Health Laboratory Service units, may contribute to the solution of some of the problems offered ; but I believe that the two together working in close and harmonious collaboration are likely to achieve far more than either of them separately. Joint research between them has only recently begun. The full exploitation of its possibilities seems to me to hold out one of the brightest hopes for the future.

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

FOOD POISONING IN ENGLAND AND WALES, 1950

An analysis of reports received by the Ministry of Health from medical officers of health, and of weekly epidemiological reports submitted by directors of constituent and associated laboratories of the Public Health Laboratory Service and by bacteriologists in certain hospital laboratories.[*]

This report has been compiled from information received from medical officers of health and laboratory directors in all parts of England and Wales. The laboratory facilities available for the investigation of food poisoning vary in different areas. Sometimes the occurrence of cases is brought to the notice of the medical officer of health and laboratory director at an early stage; at other times the investigations have to be carried out some days after the outbreak is over. The information presented therefore was not obtained under uniform conditions and it gives a general rather than a detailed account of the nature and extent of the problem. The present report resembles in form the reports already published for the period 1941–48 (Report 1950 *a*) and for 1949 (Report 1950 *b*).

Definition of terms:

an *outbreak* means two or more related cases in persons in different families,
a *family outbreak* means two or more related cases in members of the same family,
a *sporadic case* means a case which was not, as far as could be ascertained, related to other cases.
Each outbreak, family outbreak, and sporadic case has been considered as one unit and referred to in the text as an *incident*.

FOOD POISONING OF ALL TYPES

Recorded Incidence

The total number of incidents reported (Table I) was 3,979—an increase of 64 per cent. over the 2,431 incidents reported in 1949. Sporadic cases increased by 70 per cent. and outbreaks and family outbreaks by 46 per cent. The higher incidence may be real or may be accounted for, in part at least, by the fact that the new methods of reporting (Report 1950 *b*) were used for the first time for a complete year.

TABLE I

Total Incidents Reported in 1950 *by 3-monthly Periods*

Quarter	Total	Presumed Causal Agents						
		Salm. typhimurium	Other salmonellae	Staphylococci	*Cl. welchii*	Other Organisms	Chemical	Unknown
First ...	284	101	54	9	7	4	1	108
Second ...	660	314	120	20	3	11	1	191
Third ...	1,299	747	183	38	5	20	—	306
Fourth ...	760	360	83	7	9	8	—	293
Not stated	976	42	17	8	—	—	—	909
	3,979	1,564	457	82	24	43	2	1,807

The number of incidents where no bacterial or chemical cause was found increased by 933 (Table II), and the number due to salmonella infections by 652. The number of incidents due to other known causes fell from 185 to 151. The proportion of incidents due to unknown causes increased from 36 per cent. to 45 per cent. The proportion of salmonella infections fell from 56 per cent. to 51 per cent.

---

[*] The information was collated and analysed by Dr. W. Charles Cockburn and Miss E. Simpson of the Epidemiological Research Unit, The Public Health Laboratory Service.

TABLE II

Sporadic Cases, Outbreaks and Family Outbreaks—by presumed causes

Presumed Cause	Sporadic Cases	Outbreaks	Family Outbreaks	Total
Salm. typhi-murium	1,186	126	252	1,564
Other salmonellae	368	38	51	457
Staphylococci	15	45	22	82
Cl. welchii	—	24	—	24
Other organisms	3	24	16	43
Chemical	—	1	1	2
Unknown	1,415	281	111	1,807
TOTAL ...	2,987	539	453	3,979

The total number of *cases* reported to the Registrar General's Office was 7,503 but in the reports received for this analysis 17,047 persons were known to have been affected, and there were 29 outbreaks and family outbreaks where the number of cases was not stated. Of the 3,979 incidents (Table II), 2,987 (75 per cent.) were sporadic cases, and 992 (25 per cent.) were outbreaks and family outbreaks.

Salmonella typhi-murium was isolated in 1,564 incidents and other salmonellae in 457; 77 per cent. of the salmonella infections were apparently sporadic. Staphylococci were mentioned as the bacterial cause in 82 incidents, 15 of which were sporadic cases. *Cl. welchii* was isolated in 24 outbreaks and family outbreaks, and other organisms were thought to have caused 40 outbreaks and family outbreaks, and 3 sporadic cases. Two outbreaks were due to chemicals—1 to copper in tea from a defective tea urn, and 1, a family outbreak, to the contamination of porridge oats with potassium bichromate.

Seasonal incidence:—In the first quarter of the year 284 incidents were recorded; in the second, 660; in the third, 1,299; and in the fourth 760. The quarterly distribution of 976 incidents was not stated.

Vehicles of Infection

Outbreaks:—(Table III). The foods known or considered most likely to have been responsible for the spread of infection were noted in 435 outbreaks and family outbreaks (44 per cent. of the total). In some outbreaks the organism was isolated from the food and/or from the cases: in others the information was based on epidemiological observations only. In 268 outbreaks (60 per cent.) meat dishes were mentioned; 210 of these dishes were processed meats, soups, gravy, or stock. *Seventy-eight per cent. therefore of the outbreaks due to meat dishes, and* 48 *per cent. of all outbreaks in which the food responsible was mentioned were associated with processed or made-up meat such as pies, stews, sausage meat, brawn, pressed beef, reheated meat, rissoles, and cold meats.* Of the remaining meat dishes fresh meat was associated with 2 outbreaks, canned meat with 22, and " meat " which could not be further classified with 34. In most of the outbreaks due to canned meat the contents were contaminated after the can was unsealed, but in 7 outbreaks the meat was eaten as soon as the cans were opened and the evidence clearly pointed to contamination of the meat at the canning factory. Four of these outbreaks were due to *Staph. aureus;* the foods implicated were Danish canned pork, Danish canned tongue, Brazilian canned tongue and Czechoslovakian canned luncheon meat. One outbreak was due to *Salm. wien* in French canned ham. One was associated with canned luncheon meat and one with corned beef; in the two last mentioned outbreaks no pathogens were found in the meat or in specimens from the cases.

TABLE III

Outbreaks and Family Outbreaks—Vehicles of Infection and Presumed Causal Agents

Vehicle	Presumed Causal Agents							
	Salm. typhimurium	Other salmonellae	Staphylococci	*Cl. welchii*	Other Organisms	Unknown	Total	
FISH—								
Canned	—	—	2	—	—	4	6	14
Processed	1	—	—	—	—	—	1	
Shell-fish	2	—	—	—	—	4	6	
" Fish "	—	—	—	—	1	—	1	
MEAT—								
Fresh	—	—	1	—	—	1	2	268
Canned	8	1	5	—	—	8	22	
Processed and made-up	38	5	34	22	24	79	202	
" Meat "	8	1	5	—	3	17	34	
Soup, gravy and stock	1	2	—	—	1	4	8	
Trifles, ice-cream, custard, cream buns and other sweet dishes ...	21	5	6	—	5	21	58–	58
EGGS—								
Duck	38	7	—	—	—	—	45	52
Hen	2	—	—	—	—	—	2	
Dried	1	—	—	—	1	—	2	
" Eggs "	2	—	—	—	—	1	3	
MILK—								
Dried	2	—	—	—	—	1	3	16
Canned	1	—	—	—	—	—	1	
" Milk "	1	1	5	—	1	4	12	
Cheese	1	1	1	—	1	1	5–	5
VEGETABLES—								
Fresh	3	—	1	—	—	1	5	9
Canned	1	—	—	—	—	2	3	
Dried	—	—	—	—	—	1	1	
FRUIT—								
Fresh	1	1	—	1	—	1	4	6
Canned	1	—	—	—	—	1	2	
Water	—	—	—	—	—	1	1–	1
Miscellaneous (sauces, potato crisps, etc.) ...	1	—	1	1	—	3	6–	6
TOTAL ...	134	24	61	24	37	155	435–	435*

* In addition, there was one outbreak due to copper in an urn used for making tea, and one family outbreak due to potassium bichromate in porridge oats.

Fourteen outbreaks were associated with fish—6 with canned fish, probably contaminated after the cans were unsealed, 6 with shell-fish, 1 with processed fish, and 1 with fish which could not be further classified.

Fifty-eight outbreaks were associated with trifles, ice-cream, custard, cream buns and other foods of a like kind. Of the 58, 15 were associated with ice-cream and 14 with synthetic cream. With one exception, an outbreak in a hotel where 80 persons had symptoms, the ice-cream outbreaks were small and none affected more than 7 people. In 9 instances salmonellae were isolated from the cases. The hotel outbreak was due to gross contamination of the ice-cream with a variety of bacteria. In the remainder the causal agent was not found. Apart from the hotel outbreak it would seem from the small number of persons affected in each outbreak that the ice-cream was contaminated by unhygienic methods of sale to the customer. The 14 synthetic cream outbreaks were more extensive; between 2 and 200 persons were affected in each. Six were due to contamination with salmonellae, 3 with staphylococci and 2 with other organisms; 3 were due to unknown causes.

Duck eggs were associated with 45 outbreaks—38 were due to *Salm. typhi-murium* and 7 to other salmonellae. Hens' eggs and dried eggs were each associated with 2 outbreaks and " eggs " were associated with 3 outbreaks.

Milk was mentioned in association with 16 outbreaks. In the three outbreaks associated with dried milk contamination may have occurred during preparation of the feeds, and in the outbreak due to canned milk the milk became contaminated during storage after the can was unsealed. Three outbreaks were associated with T.T. unpasteurized milk. In one of these *Staph. aureus* phage Type 7 was isolated from the milk, from the dairy workers, and from the cases. In another *Staph. aureus* phage Type 42D was isolated from the milk and from one case. In the third, *Staph. aureus* phage Type 42D was isolated from the milk, but there was no record of examination of specimens from the cases. One outbreak was associated with H.T.S.T. pasteurized milk. This milk was transported in bulk prior to pasteurization. When examined after the outbreak, stained smears showed numerous cocci although culture yielded only a few *Staph. aureus*. Investigation showed that the milk might well have been contaminated from several cows, infected with phage types of *Staph. aureus* known to cause food poisoning, in one of the herds. Although pasteurization had killed the organisms the pre-formed toxin presumably was responsible for the symptoms. One outbreak was associated with milk which had been " soured by incubation " and from which *Staph. aureus* phage Type 7/42B+ was isolated. One was associated with goat's milk from a goat which later died of staphylococcal infection, and from which *Staph. aureus* phage Type 53 was isolated. One was associated with sterilized milk from which a micro-aerophilic heat-resistant bacillus was isolated, and the remainder were apparently associated with untreated milk. One of these was due to contamination with *Salm. typhi-murium*, one to *Salm. dublin* and in three the cause was not found.

Cheese was associated with 5 outbreaks, vegetables with 9, fruit with 6, and water with 1; 6 outbreaks were due to miscellaneous foods.

The causal agents in the 2 chemical outbreaks (which are not included in Table III) have already been mentioned (page 229). More information on the bacterial agents associated with the different foods is given in the table.

In 59 outbreaks the meal at which the patients were infected was known but the dish or dishes concerned were not ascertained. Eleven of these outbreaks were due to salmonellae.

Sporadic cases: The food associated with the infections was mentioned in 261 reports on sporadic cases. This small proportion (9 per cent. of the total) reflects the difficulties encountered in the investigation of sporadic cases of food poisoning, and it would be unwise to draw conclusions, from the information obtained, as to the foods mainly responsible.

TABLE IV

Sporadic Cases—Vehicles of Infection and Causal Agents

Vehicle	Presumed Causal Agents							
	Salm. typhimurium	Other salmonellae	Staphylococci	*Cl. welchii*	Other Organisms	Unknown	Total	
FISH—								
Canned	2	5	—	—	1	2	10	31
Processed	3	—	—	—	—	1	4	
Shell-fish	—	2	—	—	—	3	5	
" Fish "	8	—	1	—	—	3	12	
MEAT—								
Fresh	—	—	—	—	—	—	—	80
Canned	5	2	—	—	—	7	14	
Processed	23	13	3	—	1	16	56	
" Meat "	1	2	1	—	—	6	10	
Soup, gravy and stock	—	—	—	—	—	—	—	
Trifles, ice-cream, custard, cream buns and other sweet dishes	25	9	—	—	—	4	38–	38
EGGS—								
Duck	57	6	—	—	—	2	65	77
Hen	3	—	—	—	—	—	3	
Dried	1	3	—	—	—	—	4	
" Eggs "	5	—	—	—	—	—	5	
MILK—								
Dried	12	2	—	—	—	3	17	19
Canned	—	—	—	—	—	—	—	
" Milk "	2	—	—	—	—	—	2	
Cheese	1	—	—	—	—	1	2–	2
VEGETABLES—								
Fresh	—	—	—	—	—	—	—	1
Canned	—	—	—	—	—	—	—	
Dried	1	—	—	—	—	—	1	
FRUIT—								
Fresh	3	1	—	—	—	—	4	5
Canned	—	—	—	—	—	1	1	
Water	—	—	—	—	—	1	1–	1
Miscellaneous (beer, ginger pop, tea, bread, etc.)	3	2	—	—	—	2	7–	7
TOTAL ...	155	47	5	—	2	52	261–	261

Places where Infection Occurred: Outbreaks

The place where the contaminated food was bought or eaten was noted (Table V) in 255 out of a total of 539 outbreaks (47 per cent.). Family outbreaks were not included in this part of the analysis but many of the family outbreaks were due to food contaminated before purchase.

One hundred and four outbreaks occurred in canteens, 64 of them in school canteens; 41 occurred in hotels, restaurants and cafés; 9 in butchers' shops and 83 in hospitals, nurseries and institutions. The frequency with which other places were mentioned is given in the table.

TABLE V

Outbreaks (excluding Family Outbreaks) giving the places where the contaminated food was bought or eaten

Place	Causal Agents						Total
	Salmonellae	Staphylococci	*Cl. welchii*	Other Organisms	Chemical	Unknown	
CANTEENS—							
School	1	7	8	1	—	47	64
Works...	—	2	5	3	1	20	31
Municipal	—	1	—	—	—	3	4
Services	—	—	—	—	—	2	2
Other	—	2	—	—	—	1	3
Restaurants, hotels, etc.	11	4	1	6	—	19	41
Hospitals and institutions	41	7	4	4	—	27	83
Bakeries	—	1	—	1	—	—	2
Butchers' shops... ...	1	6	—	1	—	1	9
Cooked meat shops ...	—	1	—	—	—	1	2
Hostels and camps ...	—	—	—	—	—	4	4
Private parties	1	2	—	1	—	5	9
Bacon factory ...	1	—	—	—	—	—	1
TOTAL ...	56	33	18	17	1	130	255

The number of outbreaks traced to canteens is disturbingly high, but it should be remembered that canteen outbreaks are much more likely to be investigated in detail than are outbreaks due to the consumption of contaminated food in restaurants or from food shops. Where the infected persons disperse to their homes before the onset of symptoms—as occurs in restaurants—the tracing of the source of infection is difficult and often impossible. There is no reason to suppose that food poisoning arises less frequently in restaurants than it does in canteens. The figures reflect the difficulties of investigation of outbreaks where the persons affected may live in different administrative areas and not the relative incidence of the disease in canteen and restaurant customers.

Salmonella Infections

Infections with salmonellae comprised 2,021 incidents—51 per cent. of the total; 1,564 were due to *Salm. typhi-murium* and 457 to other salmonellae. There were 467 outbreaks—of which 303 were family outbreaks—and 1,554 sporadic cases. Fifty-five fatal cases were reported—37 due to *Salm. typhimurium*, 6 to *Salm. newport*, 6 to *Salm. enteritidis* var. *jena*, 2 to *Salm. dublin*, and one each to *Salm. cholerae-suis*, *Salm. thompson*, *Salm. bovis-morbificans* and *Salm. bareilly*. With very few exceptions the fatal cases were in persons suffering from long standing diseases such as chronic bronchitis or heart failure, or in infants and young children. The incubation period was noted in only 0·9 per cent. of salmonella incidents.

Vehicles of Infection

The vehicles of infection were noted in 158 outbreaks and family outbreaks and in 202 sporadic cases. Of the outbreaks, 64 were associated with meat dishes—43 with processed meat; 50 were associated with eggs—45 with duck eggs; 26 with trifles, ice-cream and other sweet dishes, and the remainder with a variety of fish, milk, cheese, vegetables and fruit dishes. Of the sporadic cases, 75 were associated with eggs—63 with duck eggs; 46 with meat—36 with processed meat; 34 with trifles, ice-cream etc., 20 with fish, and the remainder with a variety of foodstuffs.

Reservoirs of Infection

In 7 incidents salmonellae were isolated from poultry or animals investigated as possible reservoirs of infection in outbreaks; ducks were found to be excreters in 4 incidents, and a chicken, a pig and a dog each in one. Five outbreaks appeared to have been caused by food handlers who had had mild symptoms immediately before the outbreaks occurred, and in 6 others suspicion fell on food handlers who were found to be symptomless excreters but who might have been infected at the same time as the patients.

TABLE VI

Types of Salmonella isolated from Sporadic Cases and Outbreaks

Type	No.	Type	No.
Salm. typhi-murium	1,593	*Salm. san-diego*	1
Salm. enteritidis	26	*Salm. tennessee*	13
Salm. enteritidis var. *jena*	56	*Salm. muenchen*	1
Salm. enteritidis var. *essen*	4	*Salm. adelaide*	1
Salm. enteritidis var. *danysz*	2	*Salm. give*	2
Salm. enteritidis var. *chaco*	3	*Salm. pullorum*	1
Salm. thompson	67	*Salm. worthington*	1
Salm. newport	77	*Salm. saint-paul*	1
Salm. cholerae-suis	3	*Salm. brandenburg*	1
Salm. cholerae-suis var. *kunzendorf*	2	*Salm. manhattan*	3
Salm. bovis-morbificans	23	*Salm. menston*	6
Salm. dublin	40	*Salm. minnesota*	4
Salm. potsdam	10	*Salm. california*	1
Salm. senftenberg (*newcastle*)	3	*Salm. litchfield*	1
Salm. derby	7	*Salm. rubislaw*	1
Salm. stanley	1	*Salm. havana*	1
		Salm. pueris	2
Salm. anatum	20	*Salm. cuba*	1
Salm. reading	3	*Salm. richmond*	1
Salm. montevideo	18	*Salm. brancaster*	1
Salm. oranienburg	11	*Salm. kibusi*	1
Salm. bareilly	14	*Salm. sanga*	1
Salm. essen	1	*Salm. san-juan*	5
Salm. meleagridis	3	*Salm. wien*	3
Salm. chester	1	Unnamed and Unidentified Types	44
Salm. kentucky	3		
			2,089

The " endogenous " types are those above the horizontal line and the " exogenous " types those below the horizontal line (see text).

This table includes 68 symptomless excreters.

Salmonella Types Isolated

The frequency distribution of the different types of salmonellae isolated is given in Table VI. Similar information for the years 1923–49 was given in a previous report (Report 1950 *b*). The types above the horizontal line in the table are those which have always been common in England and Wales and may be described as " endogenous "; those below the line were seldom recognized before 1940 and may be described as " exogenous." The ratio of exogenous to endogenous types is 1 : 15 compared with 1 : 20 in 1949. This increase in the proportion of exogenous types may have resulted from the importation of these strains in foodstuffs, although it is unlikely to have been due mainly to dried eggs as were the importations in 1942 and the following years (Report 1947).

Mixed Infections

Sh. sonnei was isolated from 20 patients who were also excreting *Salm. typhi-murium*, from 3 patients excreting *Salm. enteritidis* var. *jena*, from 2 patients excreting *Salm. newport* and from 5 patients who were excreting respectively *Salm. tennessee*, *Salm. anatum*, *Salm. manhattan*, *Salm. dublin* and *Salm. pueris*.

One patient was excreting *Salm. typhi-murium* and *Sh. flexneri* Type Z, and 1 *Salm. havana* and *Sh. flexneri* Type W. A symptomless excreter was found to be infected with *Salm. typhi-murium* and *Sh. flexneri*. One patient was infected with *Salm. oranienburg*, *Salm. meleagridis* and *Sh. sonnei*.

Salm. paratyphi B and *Salm. enteritidis* var. *chaco* were isolated from 1 case, *Salm. paratyphi B* and *Salm. typhi-murium* from another, and *Salm. paratyphi B* and *Salm. brancaster* from a third.

Other Unusual Infections

In three fatal cases salmonellae were isolated from the blood stream; 1 case was due to *Salm. dublin*, 1 to *Salm. cholerae-suis* and 1 to *Salm. typhi-murium*.

Salm. cholerae-suis var. *kunzendorf* was isolated from pus from the knee joint of a child with arthritis and *Salm. montevideo* was isolated from purulent fluid from the shoulder joint in another case.

Symptomless excreters and carriers

In 120 carefully studied family outbreaks, 167 persons had symptoms and 172 were symptomless excreters.

During the year 10 persons examined routinely (e.g. food handlers, nursery entrants) were found to be excreters, and 58 persons examined as contacts of cases of other intestinal infections and for a variety of other reasons were also found to be excreters. A little information was obtained on the duration of excretion of salmonellae. One patient first examined in May, 1950, was still excreting *Salm. typhi-murium* when last examined in October, 1950. One patient known to have been an excreter in 1948 was positive when examined in September, 1950. Six persons who had had symptoms 4 weeks earlier were found to be persistent excreters and two persons were known to be excreters for 5 to 6 weeks. *Salm. tennessee* was isolated from the bile of a patient whose gall-bladder was removed.

Staphylococcal Infections

Eighty-two incidents, 2 per cent. of the total, were due to staphylococci; 15 were sporadic cases, 22 were family outbreaks, and 45 were outbreaks. The vehicle of infection was mentioned in 61 outbreaks and family outbreaks and in 5 sporadic cases. Of the outbreaks and family outbreaks 45 were associated with meat—34 with processed meat; 6 with trifles, etc., 5 with milk, and the remainder with a variety of dishes. Four of the 5 sporadic cases were associated with meat, the fifth was associated with fish.

In 55 outbreaks the incubation period ranged from 1 to 6 hours; in most the period was 2 to 4 hours.

Reservoirs of Infection

In 16 outbreaks there was evidence that the source of contamination of the food was a food handler, *Staph. aureus* of the epidemic phage type having been isolated from the hands or noses of the food handlers concerned or from boils or abrasions on the hands or forearms. Strains of *Staph. aureus* for phage typing were received by Dr. R. E. O. Williams, the Staphylococcal Reference Laboratory, Colindale, from 46 incidents. Four of the strains were untypable and 37 belonged to phage types usually associated with food poisoning. The exact significance of the remaining strains has not yet been ascertained.

Infections with Cl. welchii

In 24 outbreaks there was evidence that the cause was *Cl. welchii.* Most were due to heat-resistant strains (Dr. Betty Hobbs, personal communication) and the incubation periods in 20 of the outbreaks ranged from 6 to 18 hours, mainly from 12 to 18 hours. Twenty-two were associated with processed or made-up meat dishes. One might have been associated with potato crisps and one with jam, but the evidence in these two instances was scanty.

Incidents due to other Bacteria

Forty-three incidents were associated with organisms other than those mentioned above. Three were sporadic cases, 16 were family outbreaks and 24 were outbreaks. The incubation periods were noted in 33 incidents and they varied from one to 48 hours, chiefly from 10 to 14 hours. A variety of organisms were isolated from the foods in large numbers—aerobic and anaerobic spore-bearers, haemolytic and non-haemolytic streptococci, coliform bacilli, enterococci, *Proteus* and others.

The food contaminated with these organisms was ascertained in 37 outbreaks and 2 sporadic cases. Twenty-eight outbreaks were associated with contaminated meat—24 with processed meat; 5 were associated with trifles etc., and the remainder were associated with a variety of foods.

Incidents due to unknown causes

In 1,807 incidents (Table I) no bacteriological or chemical cause was found, often due to the delay in notification of the case or outbreak to the medical officer of health; 1,415 of the incidents were sporadic cases and 392 were outbreaks of which 111 were family outbreaks. The vehicle of infection was mentioned in 155 outbreaks and 52 sporadic cases. Of the 155 outbreaks, 109 (70 per cent.) were traced to meat dishes—79 to processed meat, 21 to trifles, ice-cream etc., 8 to fish, and the remainder to a variety of dishes (Table III). Of the 52 sporadic cases 29 were associated with meat—16 with processed meat, 9 with fish, and the remainder with a variety of dishes (Table IV). The incubation periods were noted in 210 incidents. They varied from less than one hour to 48 hours, and most occurred after an interval of 8 to 20 hours.

Discussion

The information obtained from the reports submitted by medical officers of health and by bacteriologists for the year 1950 gives substantially the same picture of the nature and extent of food poisoning as that of previous years.

The total number of incidents reported again shows an increase, some of which may be due to the fact that the new system of reporting—which came into use in 1949—has been in use for the whole year. Whether better reporting accounts for the whole of the increase is unlikely, and it appears that food poisoning is still increasing. Processed and made-up meat dishes are by far the most important foods in the spread of infection. Forty-eight per cent. of all outbreaks where the vehicle of infection was ascertained were associated with meat pies, sausage meat, brawn, cold meats, stews, reheated and made-up dishes, pressed beef, rissoles, gravy, stock, and similar dishes.

Prevention of food poisoning due to contamination of this kind of food is not primarily a bacteriological problem. If meat dishes were eaten as soon as they were cooked, the incidence of food poisoning would be greatly reduced. Those who are responsible for the preparation of food, especially meat, for consumption by the public should know that, when food is eaten as soon as it is prepared, not only is the risk of food poisoning reduced but the food is of superior flavour and palatability.

It is for the catering trade to realize their responsibilities in this matter and to devise new methods for the preparation of food in large quantity. Food prepared one or two days before it is to be eaten, allowed to cool slowly—often in a warm kitchen—and then reheated slowly to a moderate temperature well below 100° C. before it is served is potentially dangerous to the consumer. If it is impossible for meals in canteens and restaurants to be prepared and eaten within a few hours, then apparatus for the rapid cooling of the food and refrigeration at 4° C. until it is to be reheated must be brought into general use. When such food is reheated, it must be brought to boiling point before it is served.

Food poisoning is not only the result of unsatisfactory methods of preparation. Much of it is due to a disregard of elementary personal hygiene. Staphylococcal outbreaks almost always result from contamination of the food by the food handlers. As in previous years in a number of outbreaks the epidemic phage type of *Staph. aureus* has been found on the hands, in the nose, or in boils or abrasions on the face, hands, or forearms of the food handlers.

It is clear that, until food handlers are educated in personal hygiene, outbreaks will continue to occur. Hot water, soap and towels in kitchens and food preparation rooms, and the frequent use of them by the food handlers during the course of their work would make a great and immediate contribution to the solution of the problem.

When the salmonella infections are considered, the need for personal hygiene and washing of the hands after visits to the water closet is of paramount importance. Much remains to be discovered about the spread of salmonella infections, and medical officers of health and bacteriologists together could do valuable work in attempting doggedly to trace the source of infection in sporadic cases.

The need for more information on the mode of spread of salmonella infections may be illustrated by an examination of the distribution of *Salm. newport* infections in the period 1947–50 inclusive. *Salm. newport* has frequently been isolated from cases in the past, and appears as third or fourth in the list of common types. In 1947 and 1948 incidents were reported from many different areas throughout England. In 1949, 6 of the 42 incidents reported occurred in or adjacent to the West Riding; the first 2 of the 6 were extensive outbreaks, one due to the contamination of milk and one to the contamination of ice-cream. During 1949, therefore, it appears that this infection was rather more common in the West Riding than in other areas one-seventh of the total number of incidents having been reported from there. In 1950 this regional association was more definite still, and 34 of the 72 incidents recorded occurred in the West Riding, County Durham and Newcastle. One of the outbreaks in County Durham affected at least 600 persons who ate pork prepared from a sow which died of enteritis due to *Salm. newport*. But this, although extensive, was only one of the incidents, and the increased frequency of *Salm. newport* infections was evident before this outbreak occurred. It is possible that the explanation lay in a slow spread of the infection through man and animals from the two large outbreaks in 1949.

Smith and Buxton (1951) surveyed the infection rate in healthy animals and birds and isolated a variety of types of salmonellae from 2·5 per cent. of turkeys, 2 per cent. of geese, 1·2 per cent. of ducks, 0·7 per cent. of pigs, 0·7 per cent. of chickens and 0·4 per cent. of cows. They also found 1 of 500 horses to be an excreter. Previous work had shown that mice, rats, cats, dogs and pigeons may also excrete salmonellae. A sub-committee of the Staff Committee of the Public Health Laboratory Service under the chairmanship of Dr. Joan Taylor is at present investigating salmonella infection rates in ducks' and hens' eggs, in cattle brought for slaughter, in beef and pig carcasses and in healthy persons in the community. It is already clear that salmonella infections accompanied or unaccompanied by symptoms in the host are widespread in man and animals. Whatever may be shown to be the most common source of contamination of food, all foodstuffs and especially food which is not cooked and eaten hot, must be protected from the excreta of vermin, animals and birds, from flies and cockroaches, and from the excreta-soiled hands of those engaged in preparing food.

Summary

In 1950 the number of food-poisoning incidents reported by medical officers of health to the Ministry of Health and by bacteriologists to the Medical Research Council increased by 64 per cent. Of the 3,979 incidents reported, 2,987 (75 per cent.) were sporadic cases, and 992 (25 per cent.) were outbreaks and family outbreaks.

Salmonellae were the cause of 2,021 incidents, of which 1,554 were sporadic cases and 467 were outbreaks. Staphylococci caused 82 incidents, 15 of which were sporadic cases. *Cl. welchii* caused 24 outbreaks, and other organisms 40 outbreaks and 3 sporadic cases. Two outbreaks were due to chemicals, and 1,807 incidents, of which 392 were outbreaks, were due to unknown causes.

The highest number of incidents occurred in the third quarter, and the lowest number in the first quarter of the year.

The food known or believed to have been associated with the infections was mentioned in the reports of 435 outbreaks and family outbreaks; 268 of these were meat dishes—210 being processed, made-up, or reheated meat, soup or stock. *Forty-eight per cent. of all outbreaks where the vehicle of infection was mentioned were due to the contamination of processed or made-up meat.* Canned meat apparently infected at the canning factory was responsible for 7 outbreaks.

The places where the contaminated food was bought or eaten was mentioned in 255 outbreaks (excluding family outbreaks). Canteens were mentioned in 104—school canteens in 64 of these—hospitals and institutions in 83, hotels and restaurants in 41, and the remainder were in butchers' and bakers' shops, cooked meat shops, hostels, etc.

In 7 incidents salmonellae were isolated from poultry and animals examined as possible sources of infection. Five outbreaks appear to have been due to food handlers who had symptoms immediately before the outbreaks occurred, and in 6 other incidents food handlers were found to be symptomless excreters of salmonellae, but they might have been infected at the same time as the cases.

The frequency distribution of salmonella types identified in 1950 is given in Table VI.

The need for painstaking inquiries into the source of infection in sporadic cases of salmonella infection is stressed.

Renewed efforts on the part of caterers and health educators are required to inculcate the importance of personal hygiene in food handlers, the need for protection of foods from vermin, birds and domestic animals, and the necessity for changes in the methods used for the preparation of food in large quantities in restaurants and canteens.

References

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Report (1950 *a*) *This Bulletin,* **9,** 148; (1950 *b*) *Ibid.,* **9,** 254.

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SURVEY OF SICKNESS—MARCH QUARTER, 1951

(Issued from the General Register Office, Somerset House, W.C.2)

The Registrar General's Quarterly Return No. 410 (in Tables A to H) gives detailed results of the Social Survey's interviews of random samples of the adult population carried out during February to May, 1951, in which the experience of January to March was recorded. The report given here presents some rates derived from these tables without standardization or correction for the number of days in the month, and compares them with those for preceding months. More detailed studies will appear in the publications of the General Register Office.

As was explained in the previous report in this series (this Bulletin, July, 1951, p. 162) the method of drawing the sample of persons for interview was changed in February, 1951 and use is now made of local Electoral Registers instead of local records of the National Register. As a result the survey now relates only to persons 21 years and over, not 16 years and over as formerly. The rates shown for preceding periods in Tables A, B and C of this report have been adjusted to ages 21 and over, but this adjustment has not been practicable for Table D.

The " Monthly Health Index "—the proportion of persons per 100 interviewed who suffered no illness or injury so far as they remembered during the stated month—is given in Table A, separately for men and women, at ages 21–64 and ages 65 and over. There was a severe epidemic of influenza in January, 1951, and the impact of this epidemic is illustrated by the unprecedentedly low health indices given for that month. Thus successive health indices for the month of January in each year from 1947 have been as follows:—

	1947	1948	1949	1950	1951
21–64 years M	34	39	35	33	28
F	25	31	25	25	21
65 and over M	23	21	20	21	15
F	12	13	12	12	8

During February and March the health indices of men continued rather lower than in the corresponding months of previous years, but those of women returned to their usual level.

Table B gives monthly morbidity rates—the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only—distinguishing persons with illness commencing in the month from those with illness continued from the previous month. The table also gives days of incapacity reported in each month and numbers of medical consultations. Including persons with an injury the average rates for the March quarters of 1949, 1950, and 1951 compare thus:—

	Ages 21–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
Jan.–Mar., 1949	71·5	125	48·7	86·1	212	75·4
Jan.–Mar., 1950	70·2	116	49·3	84·8	215	79·0
Jan.–Mar., 1951	72·9	172	54·2	87·8	282	84·7
Jan.–Mar., 1951, per cent. of Jan.–Mar.—						
1949 ...	102	138	111	102	133	112
1950 ...	104	148	110	104	131	107

These quarterly averages indicate some increase in sickness together with a substantial increase in incapacity and in medical consultations in the March quarter 1951 compared with the two previous March quarters. However, as the monthly figures in Table B show, the increases were almost completely concentrated within the month of January, though some rates tended to be higher than usual also in February. The rates of new and recurrent illness recorded for the month of January from 1947 onwards, together with incapacity and medical consultation rates arising from all sickness, new and continued, per 100 persons interviewed, were as follows:—

	1947	1948	1949	1950	1951
New and Recurrent illnesses					
21–64 years	44	40	44	44	57
65 and over	39	39	44	42	55
Incapacity					
21–64 years	161	97	112	115	233
65 and over	255	224	217	212	371
Medical consultation					
21–64 years	49	39	48	43	60
65 and over	73	64	72	68	88

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during each month from those who developed an illness of a minor or ill-defined nature. Average monthly rates during the March quarters of 1949, 1950 and 1951 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-defined			
	Ages 21–64		65 and over		Ages 21–64		65 and over	
	M	F	M	F	M	F	M	F
Jan.–Mar., 1949	5·8	7·2	7·4	10·7	36·0	41·8	32·6	36·5
Jan.–Mar., 1950	5·3	7·3	9·1	9·5	34·9	42·3	29·6	36·0
Jan.–Mar., 1951	11·0	13·6	14·3	18·9	36·7	40·9	31·7	33·0

As would be expected under the circumstances of an influenza epidemic the increased illness reported in January, and to a less extent in the next two months, came within the category of " serious, moderate or mild." There was practically no increase in minor or ill-defined illness.

Table D gives average monthly numbers of new illnesses from three selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the numbers of days of incapacity arising from each cause (including both new and continued illness).

As mentioned above it has not been practicable in this table to adjust the rates for the younger group of adults to ages 21–64 for years 1949 and 1950, in conformity with the age-range introduced from the beginning of 1951, and the rates for 1949 and 1950 are for ages 16–64.

Acceptance of the rates given in the table as a continuous uniform series would imply an assumption that rates of illness or of incapacity at ages 16–20 were the same as at ages 21–64. Experience suggests that this is not an assumption that can reasonably be made in regard to most types of illness though it may be fairly true for influenza and colds. Going to the other extreme and assuming (again an unreasonable assumption in most cases) that the age group 16–20 experienced *no* illness, or *no* incapacity from a particular cause would lead to the conclusion that rates based on ages 16–64 would have to be increased by some 7 per cent. to make them comparable with those for ages 21–64.

Further study will be necessary to determine exactly what adjustment, between these two limits, will have to be made in each case and in the meantime it should be noted that the rates shown in Table D for 1949 and 1950 tend, probably by a small amount, to understate the illness and incapacity experienced in comparison with rates for 1951.

Rates of illness due to influenza and colds during the March quarter 1951, were the highest yet recorded in this series, with the exception of the rate for elderly women which was slightly exceeded in the March quarter, 1949. Each group showed a large increase in days of incapacity, these being roughly double what they were both in the March quarter of 1949, when there was a moderate epidemic of influenza, and in the very cold March quarter of 1947.

Large increases were also recorded in illnesses and in days of incapacity attributed to other respiratory disease, but illness and incapacity from rheumatism was not very different from that recorded during the first quarter of previous years, the only change of note being a reduction in incapacity among elderly women.

Days of incapacity from all causes, per 100 persons interviewed, by sex and age are shown in the table below:—

	Ages 21–64		Ages 65 and over	
	Males	Females	Males	Females
1949—				
January–March	122	127	200	220
April–June	86	75	151	165
July–September	82	70	89	99
October–December	98	99	225	302
1950—				
January–March	114	117	196	231
April–June	89	76	143	133
July–September	79	62	118	87
October–December	99	96	182	176
1951—				
January–March	167	177	295	271

The incapacity rate of men and women in the lower age group and of men aged 65 and over were the highest yet recorded, but the rates for elderly women (271) were exceeded three times previously, viz. March quarter, 1947 (319), December quarter, 1948 (344), and December quarter, 1949 (302).

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 21–64						Ages 65 and over					
	M			F			M			F		
	1949	1950	1951	1949	1950	1951	1949	1950	1951	1949	1950	1951
January	35	33	28	25	25	21	20	21	15	12	12	8
February... ...	34	33	31	23	26	24	18	20	16	9	11	10
March	34	37	33	23	27	26	16	20	14	12	11	12
April	39	37		26	29		19	18		12	11	
May	39	41		28	28		23	21		15	12	
June	42	42		30	30		24	21		14	12	
July	43	40		31	31		26	22		14	12	
August	43	42		31	31		26	25		16	11	
September ...	44	39		31	29		25	20		15	13	
October	39	36		26	25		24	22		14	11	
November ...	35	37		26	27		23	22		13	11	
December ...	36	34		27	26		27	20		14	12	

(Based on combined results of interviews in two following months.)

B.—*Monthly Morbidity Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 21–64				Ages 65 and over			
	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month	With a new or re-current illness starting	With illness con-tinued from previous month	Days of incapac-ity during month	Number of medical consul-tations in month
1949								
January ...	44·4	25·2	112	48	44·2	39·7	217	72
February ...	46·9	24·7	132	51	44·7	41·3	207	71
March ...	45·9	25·7	131	47	43·7	42·1	211	83
April	39·7	27·9	87	41	38·5	46·5	175	74
May	38·3	28·0	76	41	33·1	48·1	163	61
June	34·5	28·9	78	39	32·6	47·8	142	60
July	33·5	29·4	79	42	33·1	46·6	118	63
August ...	32·8	29·5	72	38	32·7	46·9	71	54
September ...	36·1	26·1	75	39	35·9	44·6	96	60
October ...	44·0	23·3	94	43	40·6	40·9	212	59
November ...	47·0	21·7	105	42	42·8	39·4	293	73
December ...	50·9	20·5	102	42	46·3	36·5	323	65
1950								
January ...	43·9	23·2	115	43	42·2	39·7	212	68
February ...	47·1	22·9	119	51	43·9	40·3	211	77
March ...	44·8	22·9	111	50	42·2	43·3	221	87
April	41·7	24·7	87	42	42·6	43·5	183	85
May	41·2	24·2	80	43	40·5	43·9	133	80
June	37·6	25·9	80	43	36·9	47·0	94	64
July	38·2	25·4	71	38	39·5	44·1	76	65
August ...	38·4	24·3	64	37	37·5	44·9	104	62
September ...	42·5	23·3	75	41	39·8	44·1	120	73
October ...	47·6	21·7	85	44	44·8	39·5	137	61
November ...	47·3	20·6	90	43	45·5	38·4	170	70
December ...	50·4	19·4	117	42	48·0	35·7	227	73
1951								
January ...	56·5	18·5	233	60	54·9	33·7	371	88
February ...	49·7	22·0	166	54	47·4	39·5	271	87
March ...	47·9	22·6	118	49	45·7	41·5	202	80
April								
May								
June								
July								
August ...								
September ...								
October ...								
November ...								
December ...								

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury, see Registrar General's Quarterly Returns, Table E.)

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 21–64		Ages 65 and over		Ages 21–64		Ages 65 and over	
	M.	F.	M.	F.	M.	F.	M.	F.
1949								
January ...	5·2	5·7	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	8·1	6·8	10·9	37·3	41·5	31·6	38·5
March ...	5·8	7·8	7·9	11·7	35·5	41·8	34·3	33·0
April	3·6	4·7	6·2	6·4	30·1	40·0	30·3	33·4
May	3·4	3·8	2·9	3·9	29·5	39·1	28·7	30·3
June	2·6	3·6	3·0	6·3	26·2	35·7	24·9	29·9
July	2·7	3·8	3·7	6·9	25·6	33·9	26·2	28·5
August ...	2·7	3·6	4·4	4·0	25·4	33·1	27·8	29·1
September ...	3·2	4·2	2·7	4·8	28·1	35·8	31·5	32·3
October ...	3·6	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·8	5·4	8·5	9·7	38·1	45·0	32·7	34·1
December ...	4·5	6·8	5·4	9·7	42·3	47·7	32·4	42·2
1950								
January ...	3·7	5·8	9·6	8·0	35·5	42·0	28·0	37·2
February ...	6·0	8·0	9·2	9·2	36·5	43·0	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·6	42·0	29·4	33·8
April	5·4	5·6	5·5	10·6	33·3	38·8	33·7	34·4
May	4·6	4·9	5·5	6·6	31·8	40·4	29·8	37·8
June	3·2	4·1	3·6	5·7	28·3	38·8	27·9	34·9
July	3·3	4·5	6·1	6·6	30·5	37·6	27·0	37·6
August ...	3·2	4·3	4·6	5·7	29·7	38·9	26·2	36·9
September ...	4·3	4·8	5·5	7·0	34·1	41·3	31·0	35·0
October ...	5·5	7·0	8·6	10·0	38·3	43·8	32·5	37·1
November ...	6·6	7·6	9·7	10·7	36·0	43·8	32·9	36·7
December ...	9·5	10·9	12·1	17·0	37·0	42·8	29·8	35·8
1951								
January ...	15·6	19·5	18·2	27·2	38·0	39·6	31·2	31·9
February ...	10·4	12·5	13·5	16·2	35·2	40·8	31·6	33·0
March ...	7·1	8·8	11·3	13·2	36·9	42·4	32·3	34·0
April								
May								
June								
July								
August ...								
September ...								
October ...								
November ...								
December ...								

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. " Ill-defined " excludes all symptomatic illness which caused incapacity for work, such cases being classed to the appropriate higher category. " Illnesses starting in each month " include new and recurrent illnesses.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity.*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 21–64*		65 and over		Ages 21–64*		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1949								
January–March... ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September... ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March... ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September... ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...	19·3	19·5	13·9	13·7	15·7	22·3	36·8	31·6
1951								
January–March... ...	22·9	22·5	17·2	18·0	67·3	81·4	78·3	103·2
April–June								
July–September... ...								
October–December ...								
	Other respiratory disease							
1949								
January–March... ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September... ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March... ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September... ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...	5·6	6·6	7·4	7·1	15·5	15·5	35·1	48·6
1951								
January–March... ...	6·4	6·7	8·5	7·9	25·3	30·2	83·5	73·5
April–June								
July–September... ...								
October–December ...								
	Rheumatism, all forms							
1949								
January–March... ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September... ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March... ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	8·2	22·2
July–September... ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...	6·5	9·1	9·3	13·1	4·7	5·8	6·3	16·9
1951								
January–March... ...	8·2	10·5	10·4	14·5	10·9	7·3	18·6	18·0
April–June								
July–September... ...								
October–December ...								

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e., new, recurrent, or continued from the previous month).

* Rates for 1949 and 1950 include persons aged 16–20.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

SEPTEMBER, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

	Sept. 1st	Sept. 8th	Sept. 15th	Sept. 22nd	Sept. 29th	Average weekly figures for Sept., 1950
Scarlet Fever	461	499	596	621	741	766
Whooping Cough	2,727	2,576	2,420	2,196	1,769	3,199
Diphtheria	35	40	36	31	25	44
Measles, excluding Rubella ...	1,877	1,388	1,211	1,069	1,258	2,816
Acute Pneumonia	217	196	212	281	258	238
Meningococcal Infection ...	36	31	23	13	23	26
Acute Poliomyelitis (Paralytic)...	35	47	42	48	64	282
,, ,, (Non-paralytic)	54	39	48	37	44	164
Ophthalmia Neonatorum ...	31	27	35	21	52	40
Puerperal Pyrexia and Puerperal Sepsis	234	231	263	246	278	74
Dysentery	152	131	103	107	144	187
Paratyphoid	37	32	38	28	33	10
Typhoid	2	4	10	10	5	6
Smallpox...	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

Correction

September Bulletin, p. 215, the telephone number of Nottingham City Hospital is now Nottingham 63361.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

AN ATYPICAL CASE OF INFECTION WITH SALMONELLA TYPHI

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Gay (1918) made the comment that " atypical cases of typhoid are particularly dangerous from the standpoint of hygiene, owing to the fact that their real condition is not appreciated." The case described below is of interest for three reasons:—the unusual site of a suppurative typhoid complication, the isolation in pure culture of a non-motile strain of *Salm. typhi* from the lesion, and the absence of any history of previous illness due to typhoid infection.

Case History

The patient, aged 72 years, the wife of a retired gardener, lived in a small house in a Shropshire village. In 1948 she noticed a swelling in the posterior triangle of her neck about 2 inches above the left clavicle. It increased slowly to the " size of a walnut " and then gradually subsided. Two years later she again noticed a swelling at the same site which eventually increased in size so rapidly that she consulted her doctor. She was referred to Mr. J. A. Baty, F.R.C.S., of Shrewsbury who found " a left supraclavicular swelling about 1½ inches in diameter—a fluctuant area with surrounding induration—swelling is in the skin and has no deep attachments ".

The swelling was provisionally diagnosed as an infected sebaceous cyst and was incised in the operating theatre under local anaesthesia after which the surgeon felt there was no reason to alter his previous diagnosis. A swab of the pus was sent to the laboratory. Numerous pus cells were seen in a direct smear and blood agar culture gave a pure growth of a Gram-negative bacillus. After biochemical tests, serological examinations were carried out against salmonella antisera. The organism, which was non-motile, was agglutinated to titre by *Salm. typhi* O antiserum but agglutination was not obtained with *Salm. typhi* H antiserum. After subculture through a Craigie tube a motile culture was obtained which was agglutinated to titre by *Salm. typhi* H antiserum. As the finding of *Salm. typhi* in an infected sebaceous cyst was so unusual, another swab was taken from the healing incision and the organism was again grown in pure culture. The patient had no history of previous T.A.B. inoculation and serological examination of her blood 7 days after operation gave the following results.

	H	O	Vi
Salm. typhi	1/100	1/100	1/80
Salm. paratyphi B	0	1/100	
Salm. paratyphi A	0		

The strain of *Salm. typhi* was sent to the Central Enteric Reference Laboratory and was found to be of Vi-phage type A.

The patient could not recall any previous illness which might have been caused by typhoid infection, and asserted that she had never spent a day in bed. Repeated specimens of faeces and urine were examined from the patient and her three relatives who lived in the same house. *Salm. typhi* was not isolated. The relatives gave no history of previous typhoid infection or of unexplained pyrexia and no suspicion of such infection was found after examination of their sera. Examination of another specimen of the patient's blood collected 5 months after the operation showed no significant change in agglutinin levels as compared with the first specimen. This suggests that she may still be harbouring the organism either in the healed skin or elsewhere. It is known that the serum of persons harbouring typhoid bacilli without excreting them usually gives a significant Vi-agglutination reaction (Felix, 1938).

There had been no cases of typhoid fever in the village in recent years, and in the absence of any other cases there was no reason to believe that there was an active source of infection in the village at this time. Routine examination of the water supply to the village did, however, show that the supply was highly unsatisfactory. Water originating from a roadside stream and said to have been chlorinated gave high faecal coli counts repeatedly, and the system of chlorination was found to be inadequate.

Discussion

Suppurative lesions complicating typhoid infection have long been recognized. Many of these originate in the bones or joints and are sometimes referred to as " silent bone abscesses." Pus formation in other sites such as the middle ear and the parotid gland are occasionally found to be due to mixed infections. Browning and Mackie (1949) state that in a certain proportion of these lesions " the typhoid or paratyphoid bacillus has been the only organism found. This has been the case in subcutaneous abscesses, in suppurative periostitis, suppuration in the parotid, abscesses in the kidneys, empyema, meningitis etc."

Ledingham and Arkwright (1912) quote a statement of Hess regarding the sites of typhoid abscesses:—" these situations have in all probability suffered some injury during the course of the initial infection, with the result that the bacillus finds there a spot of lowered resistance. It has been observed, for example, that small abscesses have formed at the site of injection of drugs like camphor and caffeine, and that the typhoid bacillus has been recovered in pure culture therefrom."

In the case described here it could be argued that the sebaceous cyst provided " a spot of lowered resistance" which led to the formation of a cutaneous typhoid abscess which was freely movable over the underlying structures.

The absence of any previous history of typhoid infection indicates that the patient may have acquired a symptomless infection in the past. Ledingham and Arkwright (1912) in referring to reports of so-called symptomless faecal excreters of *Salm. typhi* stated that " much fuller evidence is demanded before we can admit that any such cases have never at any time exhibited symptoms," and it is possible that the patient may have failed to recall an undiagnosed abortive illness. Again, it is well known that typhoid fever may take a very mild course in children and it might be difficult for a woman of 72 to call to mind a mild undiagnosed illness in childhood. On the other hand symptomless excreters of *Salm. typhi* are sometimes discovered in the course of investigation of present-day typhoid incidents. Nineteen excreters who remained symptom-free were discovered in an outbreak at an orthopaedic hospital in 1948 (Jones, 1951). It is therefore possible that the patient's failure to recall any previous illness may have been genuine and that her original infection was virtually symptomless.

Lack of motility in the strain of *Salm. typhi* on primary isolation serves as a reminder that this finding is not at all rare. This has been emphasised by Felix (1924). A motile subculture, agglutinable by *Salm. typhi* H antiserum,

was later obtained by the Craigie tube technique. Although, in this case, direct observation of the primary H inagglutinable culture gave no evidence of motility, a quotation from the Lancet (Editorial, 1946) is of interest:—
" Such has been the difficulty in deciding by direct observation whether an organism is motile that modern practice has turned to the functional test of the Craigie tube or " motility agar " in which the bacterial growth is seen to spread."

The fact that the infected cyst was incised in the operating theatre of a hospital will interest those who are concerned with the prevention of institutional cross-infection. Fortunately no secondary cases occurred in this instance but it would not be difficult to imagine a set of circumstances in a less efficient hospital whereby infection could have spread to members of the staff or patients. From time to time unexplained typhoid incidents are reported in hospitals and it is reasonable to speculate whether some of these originated from what appeared to be a relatively innocent lesion and that by the time frank cases of enteric fever had been recognized the true but unsuspected source had been discharged from the hospital.

Routine public health investigations in connection with this case showed that the water supply to the village was highly unsatisfactory owing to inadequate and irregular chlorination, and the opportunity was taken by the Medical Officer of Health to reiterate a warning to the local authority concerned of the possible consequences.

Summary

1. Isolation of *Salm. typhi* from an infected sebaceous cyst in a patient aged 72 years who gave no history of previous typhoid infection is described.

2. The strain of *Salm. typhi* was non-motile on primary isolation, but became motile after passage through a Craigie tube.

3. The unusual findings are discussed.

I wish to thank Dr. W. L. Gooch and Mr. J. A. Baty, F.R.C.S., for their observations and for permission to report this case; Dr. J. L. Gregory, Medical Officer of Health to the South West Salop Combined Districts for his valuable co-operation in the field work; Dr. A. Felix, F.R.S., for carrying out the Vi-phage typing and for his observations; Dr. D. G. Davies for his interest and collaboration in the diagnosis of the case; and Mr. P. H. Everall, F.I.M.L.T., for his efficiency in identifying the atypical strain of *Salm. typhi* from an unexpected source.

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A NEW SALMONELLA TYPE ISOLATED FROM A BABY—*SALM. NEASDEN*

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A boy aged 16 months was admitted to hospital with a history of diarrhoea and vomiting for two days. On examination the patient was found to be slightly over-weight and to have a rectal temperature of 99·4° F. He was given 5 per cent. glucose solution by mouth for 48 hours followed by increasing strength of cows' milk. The patient passed one abnormal stool immediately following admission but never vomited while in hospital. He made satisfactory progress for four days, then passed an increased number of loose motions for another four days. After the relapse the patient continued to improve and was discharged 37 days after admission.

Laboratory Investigations

A white cell count made the day following admission showed: Total W.B.C. = 14,300 (Neutrophils 38 per cent., Eosinophils 2 per cent., Lymphocytes 52 per cent., Monocytes 8 per cent.).

Faeces were cultured and an organism belonging to the *Salmonella* group was isolated on six occasions, the first being on the 11th and the last being on the 29th day of disease. Subsequent specimens of faeces failed to show the presence of the organism.

Bacteriology

This motile Gram-negative bacillus produced a colony typical of the *Salmonella* group on MacConkey and deoxycholate citrate agar. Glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose, trehalose, and inositol were fermented with the production of acid and gas. Lactose, sucrose adonitol, inulin and raffinose were not fermented after 30 days' incubation. Salicin fermentation was variable, some colonies producing acid and gas as early as 26 days, others failing to produce any reaction in 30 days. The citrate utilization test (Koser) was positive, hydrogen sulphide was produced, but indole was not formed and urea was not hydrolysed. The Voges-Proskauer test was negative and the methyl-red test positive. In gelatin at 22° C. the reaction was variable; some colonies caused no liquefaction after 22 days, whereas others produced liquefaction after 14 days.

When examined serologically this organism was agglutinated to titre by *Salm. typhi* O serum and by a serum containing only antibodies to factor IX. Absorption of *Salm. typhi* O serum by the new type removed all homologous agglutinins. A serum made with the new strain agglutinated *Salm. typhi* O to titre and absorption of this serum with *Salm. typhi* suspension removed all homologous agglutinins. Therefore, the somatic structure is represented by the symbols IX.XII.

This organism was agglutinated to titre by *Salm. dublin* H serum. When tested with monospecific sera for factors f, m, p, u, s and t, it was agglutinated by s and t. *Salm. senftenberg* H serum agglutinated the new type to titre and the new type absorbed all homologous g, s, t agglutinins from this serum. A serum made from the new type agglutinated *Salm. senftenberg* H (g, s, t) to a titre of 1/51,200 and a suspension of *Salm. senftenberg* removed all homologous H (g. s. t) agglutinins from this serum. Therefore the antigenic structure of Phase 1 is represented by the symbols g, s, t.

On testing the original culture, a few colonies were found which were not agglutinated by H (g, s, t) serum, but were agglutinated by H (e, n, x) serum. This Phase 2 was agglutinated by a monospecific serum for factor x but not by one for factor z_{15}. *Salm. abortus-equi* serum agglutinated the new type Phase 2 to titre and this organism absorbed all homologous agglutinins from this serum. Similarly a serum made from Phase 2 of the new type agglutinated *Salm. abortus-equi* to titre; this organism absorbed all homologous e, n, x agglutinins from this serum. Therefore the antigenic structure of Phase 2 is e, n, x.

Since this strain was identified, one strain has been received from a patient in Nairobi and eight strains from Cape Town isolated from an outbreak which will be reported elsewhere.

Summary

A description is given of a salmonella type isolated from the faeces of a baby suffering from diarrhoea and vomiting.

The antigenic structure is represented by the symbols IX.XII: g, s, t.–e, n, x.

The proposed name of the new type is *Salmonella neasden*.

FOUR NEW SALMONELLA TYPES FROM HUMAN SOURCES IN TANGANYIKA TERRITORY

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1. *Salm. mgulani*

A new salmonella type was isolated from the faeces of a European man aged 23 years. The patient complained of colicky abdominal pain and diarrhoea, and had a temperature of about 99·4°F. for 3 days. The duration of the illness was 7 days. The faeces were watery and contained much mucus; on microscopic examination numerous pus and red blood cells were seen.

Primary culture of faeces on deoxycholate citrate agar showed typical salmonella colonies in practically pure culture. The patient left the vicinity but was re-investigated 4 months later when no salmonella organisms could be isolated from his faeces. At this time his serum agglutinated an H suspension of the organism to a titre of $\frac{1}{50}$, but failed to agglutinate an O suspension at titres of $\frac{1}{25}$ to $\frac{1}{250}$. Immediately before his illness the patient lived at a transit camp, but no other cases of intestinal disturbance were reported at that time.

Bacteriology

The non-lactose-fermenting Gram-negative motile bacillus produced a typical salmonella colony on MacConkey and deoxycholate citrate agar. The biochemical reactions were as follows:—fermentation of glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose and trehalose with the production of acid and gas in 24 hr. Dextrin was fermented with the production of acid only in 24 hr. Lactose, sucrose, salicin, adonitol, inulin, raffinose and inositol were not fermented. Indole and urease were not formed, neither was gelatin liquefied. H_2S was produced. The V.P. reaction was negative, the M.R. test positive and citrate-utilization tests positive (Koser's and

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Simmons'). On serological examination the O antigen of the new type was identified as XXXVIII. A suspension of the strain removed all O agglutinins from *Salm. inverness* serum, and similarly *Salm. inverness* removed all O agglutinins from a serum made with the new type. The H antigens were diphasic and Phase 1 was identified as i. Reciprocal absorption tests using *Salm. typhi-murium* H (i) and the homologous serum showed complete cross-absorption between *Salm. typhi-murium* H (i) and the new type Phase 1. The second phase was tested with sera containing single factors 2, 3, 5, 6, 7 and was agglutinated by sera containing factors 2 and 3 respectively. Reciprocal absorption tests using *Salm. newport* var. *puerto-rico* H (1, 2, 3) and the homologous serum showed complete cross-absorption between the new type Phase 2 and *Salm. newport* var. *puerto-rico*.

Summary

A new salmonella type has been isolated from the faeces of an adult European suffering from diarrhoea.

The antigenic structure of the new type is represented by the symbols XXXVIII: i—1, 2, 3, and the suggested name is *Salmonella mgulani*.

2. *Salm. uzaramo*

A new salmonella was isolated from the blood and from the faeces of a young adult African suffering from an enteric-like illness.

Agglutination tests were carried out using the patient's serum and O and H suspensions of the new salmonella type with the following results:—

Day of illness	*O suspension titre*	*H suspension titre*
15	nil	nil
20	nil	nil
26	nil	1/125
29	1/25	1/250

The patient recovered and was discharged on the 29th day.

Bacteriology

The non-lactose-fermenting Gram-negative motile bacillus produced typical salmonella colonies on MacConkey and deoxycholate citrate agar. The biochemical reactions were as follows:—acid and gas were produced from glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose and trehalose. Acid was produced from dextrin and gas developed after further incubation. There was no change in lactose, sucrose, salicin, adonitol, inulin, raffinose or inositol. Indole and urease were not produced, neither was gelatin liquefied. The V.P. reaction was negative, the M.R. test positive and citrate utilization tests (Koser's and Simmons') positive; H_2S was produced. On serological examination the O antigens were identified as (i) VI, XIV, XXV. In tests with single-factor sera the organism was agglutinated by factors VI, XIV and XXV but not by I. Nevertheless an antiserum made from the new strain agglutinated *Salm.paratyphi A* O and *Salm.senftenberg* in low dilutions; conversely *Salm. paratyphi A* O and *Salm. senftenberg* O sera agglutinated the new strain in low dilutions. The new type was agglutinated to titre and removed all homologous somatic agglutinins from a serum made with *Salm. onderstepoort*. Similarly *Salm. onderstepoort* was agglutinated to titre and removed all homologous agglutinins from a serum made with the new type. Therefore the somatic antigenic structure is represented by the symbols

I, VI, XIV, XXV. It was found that the new type was monophasic and that the H phase was agglutinated to titre by *Salm. duesseldorf* H serum. Reciprocal absorption tests showed that the H phase of this organism and the new type were identical.

Summary

A new salmonella type has been isolated from the blood and faeces of an adult African suffering from an enteric-like fever.

The antigenic structure of the organism is I.VI. XIV. XXV: z_4, z_{24},—. and the suggested name is *Salmonella uzaramo*.

3. *Salm. lindi*

A new salmonella type has been isolated on two occasions from the faeces of two patients suffering from diarrhoea. The first patient was an adult European woman who complained of diarrhoea of about one month's duration. On examination she was found to have a temperature of 99° F. The second patient, an African aged 35 years, had acute enteritis, associated with abdominal pain and pyrexia. The faeces from the first patient were watery and contained a few pus cells, those from the second patient contained mucus, pus and red blood cells. In both instances the new salmonella type was not isolated from direct plates but only after culture through selenite and tetrathionate broths.

Bacteriology

The non-lactose-fermenting Gram-negative motile bacillus produced typical salmonella colonies on MacConkey and deoxycholate citrate agar. The biochemical reactions were as follows:—acid and gas were produced from glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, dextrin, rhamnose, xylose and trehalose; there was no fermentation of lactose, sucrose, salicin, adonitol, inulin, raffinose or inositol. Indole and urease were not formed, neither was gelatin liquefied: H_2S was produced. The V.P. reaction was negative, the M.R. test positive and citrate utilization tests (Koser's and Simmons') positive.

On serological examination it was found that the new type was agglutinated to titre by *Salm. inverness* O serum and that a serum made with the new strain agglutinated *Salm. inverness* O to titre. Reciprocal absorption tests of the two sera showed the O antigens to be the same; therefore the somatic antigen is represented by the symbol XXXVIII.

The new salmonella type was found to be diphasic, colonies of each flagellar phase being isolated from the original culture. Phase 1 was agglutinated to titre by *Salm. rubislaw* H (r) serum and a serum made with the new type agglutinated *Salm. rubislaw* H (r) to titre. Reciprocal absorption tests of these two sera showed the Phase 1 antigens to be the same. The second flagellar phase of the new type was agglutinated to titre by *Salm. thompson* var. *berlin* serum and by single-factor sera containing agglutinins for factors 3 and 5 but not by sera for factors 2, 6 and 7. Phase 2 of the new type removed all H agglutinins from *Salm. thompson* var. *berlin* H serum. A serum made with the new type, having a homologous titre of 1/25,600, agglutinated *Salm. thompson* var. *berlin* to titre, but absorption of this serum with *Salm. thompson* var. *berlin* suspension reduced the homologous titre to only 1/400 (1·5 per cent.). This absorbed serum also agglutinated *Salm. gaminara* Phase 2, but failed to agglutinate *Salm. bredeney* Phase 2. Hormaeche *et al.* (1944) described a " plus " factor found in some salmonella types having the second phase 1, 7. *Salm. bredeney* 1,7 does not contain the " plus " factor, whereas *Salm. gaminara* 1, 7 and *Salm. arechavaleta* 1, 7 contain this factor. The possession of the " plus " factor in Phase 2 of the new type was considered and shown to be present from the results recorded in Table 1.

TABLE 1

Salm. gaminara serum

Suspension	Titre	Absorbed new type	Absorbed *Salm. bredeney*	Absorbed *Salm. bredeney* and *Salm. arechavaleta*
Salm. gaminara 1, 7 + ...	25,600	3,200	1,600	Nil
Salm. bredeney 1, 7 ...	25,600	3,200	Nil	Nil
Salm. arechavaleta 1, 7 +	25,600	3,200	1,600	Nil
Salm. new type 1, 3, 5 +	6,400	Nil	800	Nil

Nil = no agglutination at 1/25.

Summary

A new salmonella type has been isolated from the faeces of an adult European woman and an adult African, both of whom suffered from diarrhoea.

The antigenic structure of the new type is represented by the symbols XXXVIII: r–1,3,5. The second phase of this organism also contains the "plus" factor described by Hormaeche *et al.* (1944). The suggested name for this organism is *Salmonella lindi*.

4. *Salm. matopeni*

A new salmonella type was isolated from the faeces of an adult African suffering from pyrexia, headache and abdominal pain. The illness had lasted for several weeks and a tentative diagnosis of typhoid fever had been made on the Widal reaction; the patient's serum agglutinated suspensions of *Salm. typhi* H to 1/250, *Salm. typhi* O to 1/125, but failed to agglutinate O and H suspensions of *Salm. paratyphi* A and B and the new type.

Bacteriology

The non-lactose-fermenting Gram-negative motile bacillus produced a typical salmonella colony on MacConkey and deoxycholate citrate agar. The biochemical reactions were as follows:—acid and gas were produced from glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose, trehalose and inositol; a small amount of acid and gas were formed from dextrin. No change occurred in lactose, sucrose, salicin, adonitol, inulin or raffinose. Urease and indole were not formed, neither was gelatin liquefied. The V.P. reaction was negative, the M.R. test positive and citrate utilization tests (Koser's and Simmons') positive; H_2S was produced. On serological examination the new strain was agglutinated to titre by *Salm. urbana* O serum but after absorption with the new strain the titre was reduced from 1/6,400 to 1/800 (12·5 per cent.). Similarly *Salm. urbana* was agglutinated to a titre of 1/1,600 by an O serum made with the new strain (homologous titre 1/3,200), and absorption of this serum with *Salm. urbana* left a residual titre of 1/100. Watt *et al.* (1950) described *Salm. donna* as having O antigen XXX, similar to *Salm. urbana*, but found that *Salm. donna* was unable to exhaust *Salm. urbana* serum of O agglutinins, a residual titre of 25 per cent. remaining. In view of this result serological tests were repeated using *Salm. donna* O serum, which agglutinated the new type to titre. The new type removed all somatic agglutinins from *Salm. donna* O serum; similarly *Salm. donna* O was agglutinated to titre and removed all somatic agglutinins from a serum made with the new type. The somatic antigen of the new type is represented by the symbol XXX.

The organism was found to be diphasic, colonies of each phase being isolated from the original culture. Phase 1 was identified as y and was agglutinated to titre by *Salm. tel-aviv* H (y) serum. *Salm. tel-aviv* H (y) removed all Phase 1 agglutinins from a serum made with the new strain, which was also able to remove all y agglutinins from *Salm. tel-aviv* serum. Phase 2 of the new strain was tested with monospecific sera containing agglutinins to factors 2,3,5,6,7 respectively and was agglutinated by 2 and 3; Phase 2 was agglutinated to titre by *Salm. newport* var. *puerto-rico* H serum and removed all H agglutinins from this serum. Similarly *Salm. newport* var. *puerto-rico* removed all Phase 2 agglutinins from a serum made with the new strain.

Summary

A new salmonella type was isolated from the faeces of an adult African. The antigenic formula of the new strain is XXX: y–1,2,3, and the suggested name is *Salm. matopeni.*

We are indebted to Dr. W. J. McGinniss and Dr. A. G. Poulsen-Hansen, Medical Officers, Tanganyika Territory, and to Dr. F. Padar of Dar-es-Salaam for some of the clinical details; and to the Director of Medical Services, Tanganyika, for permission to publish these findings.

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Whitehall, S.W.1.

CANICOLA FEVER IN GREAT BRITAIN

J. C. Broom, O.B.E., M.D., Wellcome Research Institution, London

Introduction

As the title indicates, this article deals with the position in Great Britain, and no attempt will be made either to give a general review of the literature, or to provide references to the large body of work on canicola fever which is being carried out in other countries, except where it bears directly on the situation here.

The first human infection with *Leptospira canicola* in this country was reported by Baber & Stuart (1946), but it seems probable that the disease was present at an earlier date, although it passed unrecognised. For example, the publication of a description of four cases of canicola fever by Laurent *et al.* (1948) reminded a medical practitioner that he had suffered from an attack of aseptic meningitis in the autumn of 1939, shortly after his dog had been seriously ill and died. Despite the interval of time, the date was clearly remembered because of its association with the outbreak of war. A specimen of serum taken in 1948 agglutinated *L. canicola* in dilutions up to 1/100, with a trace at 1/300, and reacted negatively with *L. icterohaemorrhagiae*. This evidence is not of course conclusive, but the history, symptoms, and serological findings would all accord with a diagnosis of canicola fever.

At that period the non-icteric, meningeal form of leptospirosis, which may be caused either by *L. canicola* or *L. icterohaemorrhagiae*, was seldom recognised in this country. Even now, although medical practitioners are becoming increasingly aware of its occurrence, a certain number of such cases have been brought to light almost by chance. For instance, Campbell *et al.* (1950) report that a diagnosis of canicola fever was never suspected in two patients, a husband and wife, who both suffered from an influenza-like illness, until each developed iridocyclitis in one eye. Similarly, during the investigation of samples of serum from 642 cases of aseptic meningitis, in which viral infections had been excluded, Broom (1951) detected 8 cases of canicola fever. Leptospirosis had never been considered as a possible cause of illness in any of these patients, so it seems likely that other cases may be missed, especially if the classical symptoms are absent, and there has been no special association with dogs.

The true incidence of human canicola fever is thus very difficult to assess. Broom records that 70 cases were diagnosed in England and Wales during the 4-year period 1947–50 inclusive, but a description of the first cases recognised in Scotland has only recently been published by Joe and Sangster (1951). There is, however, no evidence to indicate that it in any way approaches the figure of 3 per 100,000 of the population which Steigner and Messerschmidt (1950) consider to be a likely estimate of the annual incidence of the disease in the province of Hanover in Germany.

Epidemiology

So far as is known, dogs are the only carriers of *L. canicola* and, in these animals, the results of infection may vary from an acute, rapidly fatal illness to a symptomless carrier state. As regards the carrier state, it has been noted that marked differences occur in the degree of adaptation which has developed between different species of leptospires and their normal carrier hosts. In the case of *L. icterohaemorrhagiae* and *Rattus norvegicus* something approaching the ideal has been reached, for the parasite generally exists as a commensal, with no deleterious action on the host. In the rat, the leptospires settle in the convoluted tubules of a relatively small number of nephrons, and develop colonies on the surface of the cells lining the lumen. At intervals some of the organisms break away from the colonies, and are shed intermittently in the urine.

On occasion, however, the leptospires appear able to produce pathological effects in the rat. According to Ostertag (1950) epizootics occur among rats with up to 90 per cent. of the rodents becoming infected, and he states that he observed one wild rat, which died in captivity, showing the typical appearances of acute leptospirosis at autopsy. This must be an exceptional case, because it has often been shown that the carrier-rate among rats increases with age and, in addition, all the available evidence suggests that, once acquired, the infection persists throughout the remainder of the animal's life without shortening it.

From investigations of the epidemiology of field-fever, Popp (1950) came to the conclusion that *L. grippotyphosa* is much less well adapted to its natural host, the field-vole, *Microtus arvalis*. In one particular area where the infection rate was 80 per cent., he noted at autopsy that the kidneys of trapped voles were swollen and pale in colour. Histological examination showed severe interstitial nephritis, associated with cellular degeneration throughout the whole length of the nephron. Leptospires were present in large numbers in the glomeruli, as well as in the tubules, and the renal damage was so extensive that it seemed doubtful whether spontaneous cure would be possible.

In an attempt to obtain evidence on this point a second survey of the same area was made two months later. The vole population was then found to have decreased greatly in number and, of the 27 animals trapped, not a single individual showed leptospires in the kidneys, although agglutinins for *L. grippotyphosa* were present in the blood of 5.

During the first survey Popp had noticed that heavily infected voles were less active, and more easily caught than normal animals; taking this observation in conjunction with the apparent disappearance of carriers and the severity of the kidney lesions, he concluded that many of the animals must have died as a result of the infection.

The length of time during which these field-mice continue to excrete leptospires is not definitely known but, from these findings, Popp considers that the period is probably comparatively short.

The degree of adaptation of *L. canicola* to the dog seems to lie somewhere between these two extremes. On clinical grounds Joshua (1950) divides canine canicola fever into acute, subacute, mild, and subclinical varieties. The acute form is characterised by sudden onset, vomiting, dehydration, and relative or complete anuria. Ulceration and necrosis of the tongue and buccal mucosa appear within the first day or two of illness, and are rapidly progressive. If no treatment is given, death occurs in 36 to 96 hours from acute uraemia. This fulminating type is less common than the milder forms, which run a chronic course, and which present rather nebulous symptoms suggestive of some degree of renal damage. In the subclinical variety, infection may be present, and the kidneys heavily colonised, without any overt signs of illness.

Although it is not possible to estimate the absolute infection-rate, serological surveys in Scotland by Stuart (1946) and in England by Broom and MacIntyre (1948) show evidence of past infection in from 25 to 40 per cent. of the general canine population. Recently through the kindness of Prof. Kearney of the Veterinary College of Ireland we have been able to examine serum from 60 dogs in Dublin, and of these 17 (30 per cent.) reacted positively with *L. canicola*. It is interesting to note, however, that no human infections with *L. canicola* have as yet been recognised in Ireland.

Microscopical examination of the kidneys of healthy carrier dogs shows that the infection occurs in scattered, localised groups of contiguous nephrons. The leptospires colonise mainly the convoluted tubules and loops of Henle, where they multiply to an enormous extent without, however, invading the glomeruli or interstitial tissue. The type of infestation thus corresponds more to that seen in *L. icterohaemorrhagiae* infection of rats although, as in the sections pictured by Broom and Robertson (1951), the masses of leptospires are obviously growing at the expense of the kidney tissue. Areas of dense, round-celled infiltration can be seen, succeeded by patches of sclerosis with overgrowth of interstitial connective tissue. This interstitial nephritis appears to be progressive, and many veterinary surgeons believe it to be the cause of the chronic nephritis which is so common in older dogs.

The duration of the carrier-state in dogs is uncertain, but probably exceeds the few months which is the period generally accepted. This limit has been fixed from the results of tests made with samples of urine, and this method is not always reliable. For example, the dog which was the source of infection of the fatal human case described by Weetch *et al.* (1949) was kept under observation in our laboratory for more than a week. Its serum showed a positive agglutination titre of 1/300 for *L. canicola*, but many samples of urine were examined microscopically and by animal inoculation without demonstrating the presence of leptospires. The animal was eventually sacrificed, and a strain of *L. canicola* was isolated by injection of kidney suspension into hamsters, so the fact of its continued infection was proved beyond doubt. On microscopical examination, scattered areas of chronic interstitial nephritis were clearly visible but, despite prolonged examination, no leptospires were seen in silver-stained sections taken from a number of areas of the kidneys.

According to the husband of the deceased patient, this dog had never suffered from symptoms suggestive of leptospirosis, so no opinion can be expressed about the length of time it had remained a carrier. However, Montgomery (personal communication) states that he has demonstrated leptospires in sections of kidneys taken from dogs 4 years after clinical recovery from naturally acquired infections with *L. canicola*. In view of these findings it may be advisable to suspend judgment on the value of Popp's observations as evidence in favour of the capacity of *L. grippotyphosa* to exist over the winter as a saprophyte in surface water, as has been suggested by Kathe (1950).

Accepting the high infection rate, and assuming the lengthy carrier period in dogs, the relatively low incidence of human cases of canicola fever suggests either that the normal contacts between man and dogs are not sufficiently close to allow of transmission of the disease, or else that the leptospire has a low degree of infectivity for man. When one considers the familiarities which are often permitted to pet dogs the latter seems the more probable explanation, and the available evidence all tends to support this view. Case histories frequently show that the patient has nursed a sick dog and cleaned up its dejecta or when healthy carriers are the source of infection has mopped up pools of urine, since these animals often lack sphincter control. Such duties are usually undertaken by one person in the family, and this seems the most likely reason for the rarity of multiple cases in a household. Exceptions occur when, as in two of the cases cited by Campbell *et al.* (1950), one partner having fallen sick, the other accepts the domestic responsibilities of the ailing spouse, and thus becomes infected in his turn. In circumstances where these conditions do not obtain, we have invariably found that the other members of the household show no serological evidence of infection.

In other instances the aetiology is not so clear, but it appears probable that canicola fever, like true Weil's disease, can be contracted by bathing in contaminated fresh water. Broom (1951) records that a boy developed the infection after swimming in a quarry-hole which contained the corpse of a dog, and Joshua (1950) and Wilkins (personal communication) have both noted that dogs frequently become infected after partial or complete immersion in certain ponds.

There remain however a few cases in which no history can be obtained of contact, either direct or indirect, with infective dog urine. It may well be that a more detailed study of the circumstances in which these patients must have contracted the disease would help to eludicate some of the aetiological factors which are as yet obscure.

Symptoms and Treatment

In canicola fever, as in most leptospiral infections, there are no prodromata, and the sudden onset is ushered in with shivering or rigors, and intense headache. The temperature mounts rapidly to 103°–104°F. and the patient complains of photophobia, muscle tenderness, nausea, and vomiting. Prostration is marked, often to a greater degree than the severity of the symptoms would appear to warrant. Profuse sweating often occurs in the early stages. Signs of meningeal irritation are common, and may include mental confusion, vertigo, stiffness of the neck, and a positive Kernig's sign. Weakness, or even paresis of muscles may be present, giving rise to a suspicion of poliomyelitis. On other occasions congestion of the lungs is a prominent feature, simulating the early stages of pneumonia. In other instances abdominal symptoms predominate, and suggest a diagnosis of enteric fever. Myocarditis has occasionally been recorded, and some degree of diminution of cardiac efficiency is not uncommon. Relative bradycardia, and a reduced blood pressure are usually present.

Injection of the conjunctival vessels is found in about one-half of the cases, and is a useful diagnostic sign when present. Rashes, either morbilliform or scarletiniform in type, frequently appear, but they are often fleeting, and may not be noticed unless they are specially looked for. Jaundice is much less common than in true Weil's disease and, when present, most often manifests itself merely as an icteric or subicteric tinge.

The kidneys are generally affected, but the extent of the involvement varies widely. It may be so slight that only traces of albumin can occasionally be detected in the urine. Sometimes, however, an acute nephritis develops, and Bukh (1940) and Audoly (1948) describe cases in which the blood-urea reached 280 mg. and 576 mg. per 100 ml. respectively. Both of these patients were critically ill, but they finally recovered. In the fatal case noted above, the patient had suffered from chronic nephritis for many years, and the renal function was so badly impaired that the kidneys were apparently unable to withstand the additional strain imposed by the leptospiral infection.

The initial fever may last for a day or two only, or may continue for a week or more before the temperature falls by lysis. In either case a recrudescence of pyrexia frequently follows the afebrile period, and further relapses, though uncommon, have been reported. When the primary attack is short-lived, acute meningeal symptoms often develop with the secondary rise of temperature, and this is the form of the disease which seems most frequently to be confused with poliomyelitis.

Signs of ocular involvement, apart from conjunctival injection, may appear in the acute stages of illness, during convalescence, or even some months after recovery. Iritis, iridocyclitis, nystagmus, optic neuritis, dimness of vision, and the presence of opacities in the vitreous are among the commoner manifestations that have been reported. Fortunately the prognosis is good, and the conditions resolve without leaving residual effects.

The severe and intractable headache is the outstanding and most distressing symptom. It is quite unaffected by drugs, but marked relief is often obtained by removal of cerebro-spinal fluid, even when the tension is not increased.

Complete recovery is the rule, the only known fatalities being the case already quoted and one other which occurred in Holland in 1947, although the full report has only recently been published by Wolff, van Dam and Minkenhof (1951). Treatment is therefore essentially symptomatic but, in the presence of pre-existing kidney disease, special attention should be paid to renal function, and appropriate measures taken if the blood-urea concentration begins to rise steeply, and oliguria is marked. As Broom and Robertson (1951) point out, the damage to the kindeys is apparently reversible, and the function of the organs will be restored provided the patient can be tided over the period of danger.

The value of penicillin is difficult to assess because the disease is self-limited, and varies so widely in severity. Although there is no evidence that it has any direct curative action during the acute stages of infection, there appears to be a consensus of opinion among clinicians that the drug shortens the convalescent period, which is usually both lengthy and tedious.

Clinical Pathology

Blood

Erythrocytes. There is often a moderate degree of anaemia, the cell count falling to around 4,000,000 per cmm., and the haemoglobin value being reduced proportionately.

Leucocytes. In the early stages the total count may be normal, or somewhat diminished. Later a leucocytosis of 10–15,000 per cmm. is common, with a relative polymorph leucocytosis, but this finding is by no means invariable.

Urea. In the majority of cases the concentration is within normal limits, or is only raised to between 50 and 60 mg. per 100 ml. Of the 63 cases of which we have records, 6 showed levels of 92, 107, 120, 125, 140 and 160 mg. respectively and, in the patient who died, the concentration reached over 500 mg.

Sedimentation Rate. The E.S.R. is markedly increased from the first few days of illness, and remains high throughout the course of the illness and well into convalescence.

Cerebro-spinal Fluid

In the early stages the fluid may show no abnormalities but, by the fourth or fifth day, the protein concentration is increased, with a relative excess of globulin. The cell count also rises, sometimes reaching 2,000 or more per cmm., with either segmented or mononuclear cells predominating.

The glucose content as a rule is normal, and the importance of this estimation will be discussed later.

Urine

The urinary findings vary widely, depending on the extent to which the liver and kidneys are affected. The urine may only, from time to time, show traces of albumin; it may be loaded with bile pigments, albumin, and granular and hyaline casts; or the abnormal constituents present may represent any stage between these two extremes.

Diagnosis

Diagnosis depends primarily on the clinical findings and the history of the case, though the raised E.S.R. may help to differentiate leptospirosis from meningitis of viral origin, and a normal sugar content of the cerebro-spinal fluid is unlikely to be found in tuberculous meningitis—the two conditions with which this form of leptospirosis is most likely to be confused.

A very complete differential diagnosis has been worked out by Frey (1948) but, briefly, canicola fever should be considered in any acute febrile illness when the symptoms include meningitis, nephritis, muscle tenderness, and injected conjunctivae, especially if the history provides evidence of association with dogs.

To confirm the diagnosis, laboratory investigations are essential. The infecting organism can be isolated from the blood during the first week, or from the urine after about the twelfth day of illness. It is simpler, however, to test for the presence of specific antibodies in the serum; agglutinins first appear about the seventh day after onset of symptoms, and increase rapidly in concentration, reaching a maximum during the third or fourth week of the disease.

Leptospires are present in the blood in the early stages, and can be detected by dark-field microscopical examination of fresh specimens of blood. The cells should first be sedimented, and the supernatant fluid then centrifugalised rapidly to throw down the leptospires. In experienced hands this technique can yield valuable results, but it is impossible to reiterate too often that blood filaments and other artifacts often closely simulate leptospires in appearance, and that they may, as the result of bombardment by particles in Brownian movement, give the impression of being actively motile. It is indeed probably no exaggeration to state that the more numerous are the " leptospires " seen, the more certain it is that they are merely artifacts.

Prophylaxis

Prophylactic measures should be directed towards (1) the prevention of transmission of infection from dog to man; and (2) the elimination of the carrier state in affected dogs.

1 (*a*) An explanation should be made to any person having the care of a sick dog suspected to be suffering from leptospirosis of the risk of infection, and of the usual method of transmission. Advice should be given concerning the best methods of disinfecting and disposing of the animal's dejecta.

(*b*) As regards bathing, the precautions are essentially the same as those used for the prevention of Weil's disease. Fresh-water pools should be protected against both rats and dogs, and the water chlorinated to destroy any leptospires which may gain access.

(*c*) When a human case is diagnosed, enquiries should be made about possible sources of infection, and agglutination tests carried out on the serum of any suspect dogs. As has been noted above, apparently healthy dogs, including untrained puppies, may be symptomless carriers.

The importance of the healthy carrier is well illustrated in the case of a boy who developed canicola fever in November, 1948. Enquiry showed that a dog belonging to the family was acutely ill, and had been removed to a canine dispensary for resident treatment. Serological tests proved that the dog was infected with *L. canicola*. As the boy and the dog had been attacked about the same time, it seemed unlikely that the animal could have been the source of the patient's infection, so the position was investigated further.

It was found that, in addition to the parents, the household consisted of two boys, each of whom " owned " a dog. But the sick dog belonged to the patient's brother, who was perfectly well, and the patient's dog (a bitch) had never shown any signs of illness. However, the urine of the bitch was found to be swarming with leptospires, and the strain was cultured and proved to be *L. canicola.*

In this instance the bitch had probably been infected from some unknown outside source, and she in her turn transmitted the disease to her canine partner and her human playmate.

(2) During an attack of overt canicola fever in the dog leptospires begin to appear in the urine towards the end of the second week of illness. Joshua and Freak (1947) found that administration of penicillin produced clinical cure of the disease, but Brunner and Meyer (1949) showed that this antibiotic did not eliminate leptospires from the kidney, and that dogs so treated continued to shed the organisms in the urine. They proved, however, that a subsequent course of streptomycin, in doses of 40 mg./Kg. body-weight daily for four days, would effectively terminate the carrier state. Uhlenhuth and Schoenherr (1951) also noted that treatment with convalescent serum was clinically effective, without, however, preventing colonisation of the kidneys and the development of a carrier state. These workers made an intensive study of the action of antibiotics in hamsters experimentally infected with a strain of *L. canicola* which invariably proved fatal in untreated animals. Their results showed that, whereas chloramphenicol was completely inactive, aureomycin and terramycin (particularly the latter) effected clinical cure, even at a relatively advanced stage of the disease, and also prevented residual infestation of the kidney. Their experiments were not extended to include the treatment of carrier dogs, and this is a necessary corollary before these two drugs can be confidently used in the treatment of canine canicola fever as a prophylactic measure in the prevention of the human disease.

Summary

1. Between fifteen and twenty human cases of canicola fever are recognised annually in Great Britain, but evidence suggests that the true incidence is probably higher.

2. Although *Leptospira canicola* is less well adapted to the dog than is *L. icterohaemorrhagiae* to the rat, dogs probably remain carriers for years, and so continue to act as a reservoir of infection.

3. Transmission of *L. canicola* to man is most often effected by direct contact with infected dog-urine, but it can also be brought about by bathing in contaminated water.

4. A short review is given of the symptoms, methods of diagnosis, and treatment of human canicola fever.

5. Appropriate measures to prevent transmission of *L. canicola* from dog to man are described, and also the methods which should be used to eliminate the infection in carrier dogs.

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NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

OCTOBER, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

	October 6th	October 13th	October 20th	October 27th	Average weekly figures for October, 1950
Scarlet Fever	869	1,032	1,135	1,116	1,156
Whooping Cough	1,589	1,612	1,516	1,441	2,666
Diphtheria	36	41	33	35	40
Measles, excluding Rubella ...	1,466	1,648	1,809	1,962	5,358
Acute Pneumonia	310	395	424	419	400
Meningococcal Infection ...	27	35	37	35	30
Acute Poliomyelitis (Paralytic)...	56	54	67	62	211
,, ,, (Non-paralytic)	40	40	52	34	87
Ophthalmia Neonatorum ...	45	37	41	30	35
Puerperal Pyrexia and Puerperal Sepsis	241	251	245	207	79
Dysentery	124	118	162	151	194
Paratyphoid	31	90	81	29	12
Typhoid	9	6	3	4	6
Smallpox	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Bournemouth Laboratory

The Public Health Laboratory at Poole, under the direction of Dr. G. J. G. King, moved early in October to new quarters in Bournemouth. The new address is: Public Health Laboratory, Gloucester Road, Boscombe, Bournemouth (*Tel:* Boscombe 37304).

Bedford Laboratory

A new laboratory has now been opened in Bedford, under the direction of Dr. W. F. Lane. The address is: Public Health Laboratory, General Hospital, Kimbolton Road, Bedford (*Tel:* Bedford 66896).

Harrogate Laboratory

A constituent unit of the Public Health Laboratory Service was established in the Harrogate and District General Hospital in October. The director is Dr. L. A. Little, and the address is: Public Health Laboratory, Harrogate and District General Hospital, Harrogate (*Tel:* Harrogate 83221).

Middlesbrough Laboratory

A constituent unit of the Public Health Laboratory Service was established in Middlesbrough General Hospital in October. The director is Dr. A. R. Blowers, and the address is: Public Health Laboratory, General Hospital, Ayresome Green Lane, Middlesbrough (*Tel:* Middlesbrough 89191).

REGIONAL CENTRES FOR THE LABORATORY DIAGNOSIS OF LEPTOSPIRAL INFECTION

In view of the large number of specimens now being sent for diagnosis to Dr. J. C. Broom at the Wellcome Laboratories for Tropical Medicine, London, N.W.1, the Public Health Laboratory Service have arranged, at his request, for preliminary screening tests to be carried out at a number of their laboratories in different parts of the country. Difficult sera, or sera giving doubtful results, will continue to be referred to Dr. Broom. The new arrangements will come into operation on 1st November, 1951.

Pathologists who would like to take advantage of this may now send material to the nearest of the laboratories on the list given below:—

Place	*Address*	*Pathologist in charge*	*Telephone Number*
Birmingham	Public Health Laboratory, 150 Great Charles Street, Birmingham, 3.	Dr. B. R. Sandiford	Central 6921/2
Cambridge ...	Public Health Laboratory, Tennis Court Road.	Dr. R. M. Fry ...	Cambridge 55526
Exeter ...	Public Health Laboratory, 7 Dix's Field.	Dr. B. Moore ...	Exeter 54959
London ...	Central Public Health Laboratory, Colindale Avenue, N.W.9.	Lt.-Col. H. J. Bensted.	Colindale 6041 & 4081
Newcastle-on-Tyne.	Public Health Laboratory, General Hospital, Westgate Road, Newcastle-on-Tyne 4.	Dr. A. I. Messer ...	Newcastle 34920
Portsmouth ...	Public Health Laboratory, Block "G", Infectious Diseases Hospital.	Dr. K. E. Hughes ...	Portsmouth 74785/6/7
Cardiff ...	Public Health Laboratory, Institute of Preventive Medicine, The Parade.	Dr. Scott Thomson	Cardiff 8288

THE LABORATORY DIAGNOSIS OF VIRUS INFECTIONS OF THE CENTRAL NERVOUS SYSTEM

F. O. MacCallum, M.D.

Virus Reference Laboratory, Colindale, London, N.W.9.

In a previous paper (MacCallum, 1947) a description was given of the collection, storage and mode of transmission of specimens taken from patients suspected of suffering from virus disease, and of the kinds of examination that could be usefully carried out on them. The present paper is devoted to the diagnosis of virus infections of the central nervous system.

Main Principles and Methods

Tissues of the nervous system can react in only a limited number of ways to infection so that, apart from rabies and classical poliomyelitis with flaccid paralysis of limbs, it may be very difficult to make a specific diagnosis except by virus isolation or serological tests. The tests for some of the known viruses that infect the central nervous system are in the stage of development and there are a number of presumed virus infections in which the aetiology is still being investigated. The types of illness in which the laboratory can be of help or would like material for investigation are described below.

Laboratory diagnosis is made (*a*) by isolation of the virus from body fluids, secretions, excretions or tissues by means of inoculating fertile hens' eggs or animals, or (*b*) by the demonstration of a rising titre of complement-fixing or neutralizing antibodies in the blood in convalescence. The first process is much more expensive and time-consuming than the latter, seldom gives an earlier positive answer, and must in any case be confirmed by serological tests. Therefore, the examination of serum, usually for complement-fixing antibodies, is the method of choice for routine diagnosis; serum neutralization tests are performed in special circumstances. Two samples of serum from each patient are essential, one taken at the onset of illness or when the patient is first seen, and the second usually during the third or fourth week. The laboratory can give little or no help if the patient is first seen after the third week of illness. Virus isolation is usually only attempted in localized outbreaks or in special circumstances described below.

If in doubt, specimens should always be collected in the acute stage and stored in the ice-box, and the laboratory communicated with.

Although the results are usually not available until the patients have recovered, they are valuable from the standpoint of public health and epidemiology as well as for completing the diagnosis of the individual patient.

Meningitis

Perhaps the largest group of cases comes under the heading benign lymphocytic meningitis. It is now recognized that in practically all virus infections the meninges may be affected. In most instances this can only be detected by finding an increase in cells and protein in the cerebrospinal fluid, but in some cases clinical evidence of meningitis is obvious. When this accompanies or immediately succeeds a recognized virus infection such as mumps or hepatitis, the diagnosis is obvious; but when it occurs a month or more after or is completely unrelated to such an illness, laboratory tests may be of value. Viruses that can be tested for in this group are lymphocytic chorio-meningitis (L.C.M.), mumps, herpes simplex, lymphogranuloma venereum-psittacosis group, louping-ill, and Coxsackie (C) viruses. There are no routine tests for poliomyelitis virus. Infection with *Leptospira*, *Toxoplasma* and, of course, the tubercle bacillus may produce a similar clinical picture. Tests to date indicate that in England the organisms mentioned above account for at most about 20 per cent. of the non-tuberculous cases; the remainder are caused by either some as yet unrecognized viruses or are examples of infection with poliomyelitis viruses.

Collection of specimens

For investigation of cases of lymphocytic meningitis the following specimens should be obtained:—

A. *On admission.* (i) 10 ml. of blood collected in a sterile dry syringe and placed in a clean dry sterile bottle. The serum should be separated from the clot within 24 hours of collection and kept at 4° C. or lower. Unless the clot is needed for special virus investigations it may be discarded. The serum may be sent by post.

(ii) 5–10 ml. of cerebrospinal fluid in a sterile container with a rubber bung or screw cap. The cerebrospinal fluid should be frozen immediately on collection (the freezing chamber of a domestic refrigerator is satisfactory), and must be despatched to the laboratory packed in ice or, preferably, carbon dioxide snow (dry ice). Bottles should not be filled more than two-thirds full in order to prevent breakage by expansion of liquid on freezing.

B. *In convalescence.* A second sample of serum from 10 ml. of blood at the 14th to 21st day from the onset. The serum should be sent by post.

In general, sera should be collected when a patient is first seen and again between the 14th and 21st day. If patients are being discharged before this time the second sample should be collected just before departure. A negative result for mumps or *Leptospira* after the 14th day of disease is usually significant, but not until after the 21st day in L.C.M. Antibodies for mumps and Coxsackie viruses may appear within 7 days of the onset of illness and moderately high titres may persist for several years, so that a rising titre must be obtained in order to interpret the significance of the result in relation to the present illness. The maximum titre of complement-fixing antibodies for L.C.M. is usually not high (1/2 to 1/32) and, as the mild prodromal influenza-like illness may persist for several weeks before the signs of meningitis occur, the serum may already be positive when the patient is first seen with meningitis. Antibodies may persist for 6–12 months, so here also a rise in titre must be demonstrated in successive specimens.

The antigens used are made from fertile hens' eggs or animal tissues such as guinea-pig spleen and mouse brain. As certain sera may react with non-infected tissues, these are always used as controls in tests, and a four-fold difference at least between the result with normal and positive antigen is necessary to render significant the result on a particular serum.

Titres of serum in complement-fixation tests

(a) *Lymphocytic choriomeningitis*

A persistent positive titre of 1/2 is seldom seen in L.C.M., but this low titre may be significant in a second specimen if the first specimen is negative, and especially if this titre persists for several weeks and later becomes negative. The usual titres are 1/8 to 1/16 but may be higher, and the complement-fixing antibodies disappear after 6–12 months. Occasional false positives have been described in patients with brain tumour. If there is any doubt, confirmation may be obtained by recovery of the virus from the brains of mice trapped in the patient's home, and tests on serum of other members of the household. The latter are, however, frequently negative.

(b) *Mumps*

Two different antibodies appear in the serum following infection with this virus. One reacts with V (virus) antigen and one with S (soluble) antigen. The S antibodies begin to rise earlier, about the 5th day, never reach such a high titre, and disappear sooner, than the V antibodies, which do not begin to rise till about the 7th to 10th day. A significant titre of anti-V is 1/40 or higher; but a titre of 1/40 should be regarded as significant only if it represents an increase from zero and the controls are negative, because a titre as high as 1/80 or 1/160 may persist for a year or more. The usual titres are 1/160 to 1/640.

In a case of lymphocytic meningitis with its onset a month after parotitis and an unchanging titre of 1/160, it may be impossible to make a specific diagnosis, especially if it occurs in the poliomyelitis season; an example of such a case occurred in the summer of 1950. On the other hand, cases of lymphocytic meningitis in the poliomyelitis season may be mumps infection without parotitis.

Some examples of laboratory findings in cases of lymphocytic meningitis are shown in the table.

TABLE

Some examples of laboratory findings in cases of lymphocytic meningitis

	Type of illness	Days since onset of present illness	Mumps		A/C	Month of Year
			Titre V	Titre S		
Case 1	Meningitis with no previous history of parotitis	17	1/320–1/640	1/160	1/40	July/ Aug.
		94	1/640	1/80		
	Diagnosis—mumps meningitis					
Case 2	Parotitis 7 days previous to meningitis	8	1/160	1/40	—	Oct.
		31	1/640	—		
	Diagnosis—mumps meningitis					
Case 3	Parotitis 16 days before onset of meningitis	10	1/320	1/40	—	Aug.
		22	1/320	1/40	—	
	Diagnosis—Possibly mumps meningitis but could be poliomyelitis or other unknown virus meningitis succeeding mumps infection					
Case 4	Parotitis several months previous	3	1/80	1/20	1/10	Aug.
		30	1/80	1/20	1/10	
	Diagnosis—not mumps meningitis					
Case 5	Parotitis 6 months previous	7	1/160	1/20	—	Aug.
		21	1/160	1/20	—	
	Diagnosis—not mumps meningitis					

V = virus antigen of mumps
S = soluble antigen of mumps
A/C = normal antigen control titre

Coxsackie viruses

Knowledge of infections with this group of C viruses is at the present moment limited and routine investigation of single cases for the virus or complement-fixing antibodies is not practicable. At least thirteen immunologically distinct types have been described in the U.S.A., and evidence of the presence of three of these types has been obtained in England. Infection with two types of the C virus has been described in the same family and three viruses in a community at the same time. The presence of both C viruses and poliomyelitis viruses in the same patient has been described on a number of occasions. Some patients have shown a rise in titre to both a C virus and poliomyelitis virus during an attack of an apparently single disease. As complement-fixing antibodies to some types of C virus may appear within the first seven days of illness, it is essential to obtain a specimen of serum immediately the patient is seen and to demonstrate a rise in titre later. Titres appear to be 1/10 to 1/80 in recent infections. An unchanging titre of 1/10 or 1/20 cannot be considered significant in relation to the illness under investigation.

Poliomyelitis

Cases of obvious poliomyelitis are only investigated in order to obtain strains of virus for typing or when there is a possibility of obtaining epidemiological information which may be of value for a better understanding and control of the disease.

Poliomyelitis virus infection can be identified only by isolation of the virus from the stools or pharyngeal washings during life or from the spinal cord of fatal cases by inoculation into monkeys. Only one (Lansing) type of the three or more immunological types is infective for mice. At the present time only serum-neutralizing antibodies can be estimated and these only by inoculation of virus-serum mixtures into monkeys with two types and mice with the third type. Due to insufficient multiplication of the virus in animal tissues, complement-fixing antigens have not been available.* Recent observations on the growth of the virus in tissue cultures of human embryonic tissues indicate that there may be a more simple *in vitro* immunological test in the near future.

Encephalitis

The only arthropod-borne encephalitis virus infection known in Great Britain is louping-ill. This is a tick-borne disease of sheep which has been recognized only in certain areas in Scotland and the northern border counties of England and possibly certain areas of Wales. Natural infection of man has recently been reported in the Northumberland area and Scotland, but the frequency of human infection is not known. A complement-fixing antigen is available and investigations of individual patients and population surveys are being carried out.

Other cases of acute non-bacterial infective encephalitis, unrelated to recent recognized acute infections such as measles, influenza or zoster, may be caused by the virus of herpes simplex and possibly mumps virus, but this latter usually produces a meningo-encephalitis. Still other cases of clinical encephalitis of this type may be caused either by as yet unidentified viruses or by a poliomyelitis virus.

As in virus meningitis, the serological tests are the most satisfactory as a routine in encephalitis investigations. However, virus may be isolated from the cerebrospinal fluid and sometimes from the blood of patients in the acute stage of encephalitis, and if a group of cases occurs in a household or community it is desirable to try and isolate the virus from the blood (10·0 ml.) or cerebrospinal fluid (5–10 ml.). Stools (15–20 gm.) should be collected from the same patient so that they can be examined for poliomyelitis virus if the other tests are negative. Specimens should be collected, stored and transported as described for meningitis.

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* Since this paper went to print, Casals and his colleagues have described the preparation of a complement-fixing antigen from the C.N.S. of new-born mice infected with the M.E.F.I. strain of virus (Lansing type). This antigen also appears to give some overlapping with sera of convalescents from infection with the two other main types of poliomyelitis virus (Brunhilde and Leon), so that it may be of great practical value in the future. (Casals, J., Olitsky, P.K., and Anslow, R.O. (1951), *J.exp.Med.*, **94**, 123.)

In rapidly fatal cases, rectal contents (15–20 gm.) and pieces of motor cortex, cerebellum, pons, medulla and cervical cord 1 cm. square, should be placed in 50 per cent. neutral buffered glycerin or 50 per cent. glycerine-saline, stored at 4° C. and transported to the laboratory on ice. Of course, brain and cord should also be examined histologically.

Encephalitis lethargica

Acute encephalitis lethargica, which was epidemic in the period 1918–27, seems to have almost disappeared. Its aetiology was never satisfactorily determined although herpes simplex virus was isolated from the cerebrospinal fluid or brain of a few patients. The acute disease is still diagnosed from time to time and it is important that the aetiology of such cases be investigated whenever possible.

Specimens should be collected and submitted in the same way as for acute encephalitis (above).

Post-infectious encephalitis

The present view is that this disease represents an allergic response of the central nervous system to specific virus, i.e. measles, zoster, etc. The cases are generally seen too late for useful tests to be carried out.

Post-vaccinal encephalitis

The aetiology of this group is again considered to have an allergic basis. Acute deaths following vaccination are fortunately very rare; but if death occurs within 4 to 5 days of the onset of symptoms of encephalitis, investigations into the aetiology should be carried out. Brain and cord should be examined histologically and pieces of brain, cord and faeces should be collected and frozen (see encephalitis). Virus investigations will be carried out when the results of the histological examinations are known.

Acute infective polyneuritis

(Guillain-Barré's syndrome—Landry's Paralysis)

Attempts to isolate a specific virus from patients with this illness have been unsuccessful, although there has been a report that a Coxsackie virus was recovered from the stool of one case in Toronto, Canada, and an agent with an apparently low pathogenicity for monkeys and producing a pathological picture of polyradiculitis in them was recovered from the stool of a child thought to be suffering from acute anterior poliomyelitis in Boston, U.S.A.

Sera should be collected at the onset and about the third week.

If a number of cases occur in a community it may be worth while to collect spinal fluid and stools from living cases and rectal contents, cord, ganglia, and affected peripheral nerves from rapidly fatal cases, for virus studies.

Acute disseminated encephalomyelitis

The possibility of laboratory investigations in such cases should be discussed directly with the laboratory when they occur.

Encephalomyocarditis

Infection with viruses known as the E.M.C. group has not yet been found in Great Britain, but if present it should be brought to light by the tests described above.

Reference

MacCallum, F. O. (1947) *This Bulletin*, **6**, 229.

AN INVESTIGATION INTO THE INCIDENCE OF *SALM. DUBLIN* IN HEALTHY CATTLE

J. Morrison Ritchie, M.A., M.B., D.P.H., Public Health Laboratory, Birkenhead, and N. M. Clayton, M.R.C.V.S., F.R.San.I., Municipal Veterinary Officer, Birkenhead.

In November, 1949, the Salmonella Sub-Committee of the Public Health Laboratory Service decided that, as an extension of the research into the salmonella carrier rate in the human population, a similar investigation should be made in domestic animals. Birkenhead is one of the largest cattle-slaughtering centres in the country, and the local laboratory instituted a joint investigation with the Birkenhead authorities into the salmonella carrier rate in healthy cattle. There are two establishments, the Lairage (Foreign Animals Wharf) containing storage space and rest houses for animals, with slaughtering facilities, and the Birkenhead Corporation Abattoir which is used for the slaughter of cattle from this country. One of us (N.M.C.) is in charge of the meat inspection in both places. Samples of faeces from individual beasts before slaughter, and of bile and liver after slaughter, have been examined for the presence of salmonellae.

Collection of Samples

1. *Faeces*

(*a*) Cattle pens were cleaned scrupulously by scrubbing with clean water to exclude any possible carry-over of infection from a previous beast.

(*b*) Each animal was given a thorough veterinary examination, and any beast not classified as completely sound clinically was excluded from the investigation. Even if an animal passed the clinical test, it was rejected if its faeces were not of normal appearance and consistency. These veterinary precautions were regarded as an absolutely essential part of the investigation.

(*c*) For sampling, an ordinary faeces pot and spoon were used, and all specimens were taken personally by one of us (N.M.C.). The top layer of the pile of faeces was displaced by the back of the spoon, which was dug well into the centre of the faecal mass, and the sample withdrawn. Splash contamination is a danger in work of this kind, but its effects can be reduced to a minimum by careful technique.

2. *Bile*

Samples of bile were taken and placed separately in 1 oz. screw-cap containers. The samples were taken immediately after the slaughter of the animal.

3. *Liver swabs*

Healthy livers were incised, any diseased or inedible livers being discarded. One incision only was made in each liver. With ordinary throat swabs in separate test-tubes, the cut surface was swabbed over, and each swab was cultured separately in the laboratory.

Laboratory Investigations

Initially, primary culture was made in both selenite and tetrathionate broths, but as selenite broth was found to give slightly better results this was later used alone. Direct plating was tried but discarded as ineffective owing to the large number of paracolon bacilli found. After 24 hours' incubation the selenite broth culture was inoculated on to plates of MacConkey, deoxycholate, and Wilson-Blair agar. Considerable trouble was encountered with paracolon bacilli in the early stages, but was avoided later by the use of deoxycholate plates with the addition of sucrose. Non-fermenting colonies on lactose-sucrose-deoxycholate plates were cultured in peptone water with addition of dulcitol. Dulcitol fermenters were cultured in glucose, lactose, sucrose, mannitol and salicin as a routine. Some strains of *Salm. dublin* not fermenting dulcitol for 48 hours were found. A few anaerogenic strains of this organism were also met with; some of these strains occasionally produced gas on further incubation, but most failed to do so.

Strains giving salmonella fermentation reactions were tested with polyvalent salmonella serum issued by the Standards Laboratory, Colindale. Those giving positive slide agglutination were then tested with *Salm. dublin* H serum, and those failing to react were further tested with *Salm. typhi* O serum (for *Salm. dublin* O). As *Salm. dublin* was almost the only *Salmonella* isolated, only those organisms not agglutinated by this serum were tested with any other sera. One or two strains of *Salm. dublin* were in the non-motile form when isolated.

Initial positive slide agglutination was always confirmed by further tests and no strain giving only doubtful positive slide agglutination proved to be a *Salmonella*. This observation greatly simplified the investigation. The identity of all strains of *Salmonella* isolated was confirmed by the Salmonella Reference Laboratory.

Findings and Discussion

During the period from 1st November, 1949, until 31st December, 1950, 2,109 samples of faeces and 334 of bile were examined. Of these, one sample of faeces from a cow originating in the Crewe area yielded *Salm. typhi-murium*. Otherwise the only salmonella type found was *Salm. dublin*, which will be considered exclusively henceforth. The results are given in Table 1.

TABLE 1

Faeces and bile from individual cattle yielding *Salm. dublin*

	Faeces (separate beasts)			Bile (separate beasts)		
	Total	Positive	Percentage	Total	Positive	Percentage
Irish	1,254	126	10·0	116	4	3·4
Non-Irish ...	855	32	3·7	218	1	0·5

The possibility of cross-contamination of samples was investigated, but it was believed that the technique for their collection had practically eliminated this.

Conditions of transport

The Irish animals investigated were chiefly bullocks. These may have been in transit together for some days before samples were taken, and the possibility of cross-infection during this time cannot be ruled out. If cross-infection did take place it was insufficient to cause clinical symptoms before slaughter.

Non-Irish animals were bullocks and cows, the bullocks being in the same transport category as the Irish. Most of the non-Irish beasts were older, consisting mainly of throw-outs from herds. These animals came from cattle sales in the neighbourhood, and in any batch of cattle several may have come from an infected herd and been in contact with each other for weeks. This may have resulted in a high figure for one particular batch, and a misleading indication of the proportion of cattle infected. For example, one consignment of old cows from a particular area in Cheshire yielded 13 positives out of 20, whereas no other batch from that point yielded any salmonellae at all. From another batch of old cows 12 specimens of faeces were examined; *Salm. dublin* was isolated from seven cows and *Salm. typhi-murium* from one. Though each of these animals was sound on clinical examination, there must have been some reason why the animal was discarded.

It was decided therefore to record the findings in terms of individual samples of faeces, and also as batches of individual samples, where the word " batch " was used to denote a number of samples of cattle faeces collected at the slaughterhouse at one time. The number of samples in a batch necessarily varied with the supply of animals. It was usually about 10 or 12, but might be as high as 20 or as low as 2 or 3. There was no pooling of specimens in a batch, and each specimen was examined separately throughout.

TABLE 2

Batches of cattle faeces yielding *Salm. dublin* from one or more individual animals

	Faeces (batches)			Bile (batches)		
	Total	Positive	Percentage	Total	Positive	Percentage
Irish	216	108	50·0	22	3	13·6
Non-Irish ...	57	9	15·8	27	—	0·0

Note.—In this and in subsequent tables the term " batch " refers to samples of faeces or bile collected from the slaughterhouse at one time and examined individually.

It is noteworthy that, over a period of 14 months, from 50 per cent. of batches of faecal specimens taken from Irish cattle *Salm. dublin* was isolated from one or more animals. Though it is of interest to know the percentage of batches found to be infected on reaching the abattoirs, the main object of the investigation was to find the carrier rate in the animals for slaughter. It will be seen from Tables 1 and 2 that the proportion of Irish to non-Irish cattle excreting *Salm. dublin* in the faeces is similar whether individual cows or batches are considered.

Table 3 distinguishes between bullocks and older beasts among the non-Irish cattle.

TABLE 3

Non-Irish cattle faeces yielding *Salm. dublin*, and batches of faeces yielding *Salm. dublin* in one or more batches

	Faeces (young cattle)			Faeces (old cattle)		
	Total	Positive	Percentage	Total	Positive	Percentage
Number of specimens ...	174	4	2·3	681	28	4·1
Batches ...	14	2	14·3	43	7	16·3

The young non-Irish cattle were chiefly bullocks, and had been in contact during transport for periods of up to a few days only. The figures of 2·3 per cent. and 14·3 per cent. in the young cattle of Table 3 can therefore be strictly compared with the Irish bullock figures of 10 per cent. and 50 per cent. in Tables 1 and 2, as the period of contact during transport was much the same in each case.

Both in Ireland and in this country there are considerable differences in the area incidence of *Salm. dublin*. It has been assumed that the Irish ports of exit represent collecting centres serving specific areas from which cattle are sent. Tables 4 and 5 have been prepared to show the percentage incidence in the different areas of Ireland and this country respectively. The distinction between individual faeces percentages and batch percentages has been maintained. Limerick, though not a port, has been included as an area, as animals were known to have come from there. It will be seen that no Irish area proved to be free from infection.

TABLE 4

Area incidence (Irish) of *Salm. dublin* in cattle faeces

	Cork	Dublin	Drogheda	Waterford	Limerick	Belfast
Number of specimens	220	639	107	148	55	85
Positive	18	65	4	12	10	17
Percentage	8·2	10·2	3·7	8·1	18·2	20·7
Batches	23	68	6	19	6	10
Positive	12	34	4	5	4	7
Percentage	52·2	50·0	66·7	26·3	66·7	70·0

TABLE 5

Area incidence (non-Irish) of *Salm. dublin* in cattle faeces

	Cheshire	Denbigh	Flint	Anglesey	Lancashire	Scotland
Number of specimens	418	181	90	16	82	55
Positive	23	0	5	0	1	3
Percentage	5·5	0·0	5·6	0·0	1·2	5·5
Batches	19	14	13	1	7	7
Positive	4	0	3	0	1	1
Percentage	21·0	0·0	23·1	0·0	14·3	14·3

Many of the animals noted in Table 5 were throw-outs from dairy herds and in some instances a batch yielded an abnormally high number of positives. All the 23 positive samples of faeces from Cheshire, for example, came from two markets, while from 241 dairy cattle on farms examined in a collateral investigation not one sample of faeces yielded *Salm. dublin*.

The variation in seasonal incidence of *Salm. dublin* in the faeces of the Irish cattle was less than was expected. Table 6 analyses the seasonal distribution.

TABLE 6

Seasonal incidence of *Salm. dublin* in the faeces of Irish cattle

	Number of specimens	Number of positives	Percentage
Nov.–Dec., 1949	85	16	18·8
Jan.–Feb., 1950	197	24	12·2
Mar.–Apr., 1950	137	19	13·9
May–June, 1950	232	25	10·8
July–Aug., 1950	181	18	9·9
Sept.–Oct., 1950	249	20	8·0
Nov.–Dec., 1950	173	4	2·3

There is an unaccountable fall in the number of isolations for the period November–December, 1950. Possibly there may have been an epidemic of *Salm. dublin* in Ireland which was on the wane when the investigation began, or possibly there was some seasonal variation which was masked by an abnormally high rate during the winter of 1949–50. It is, however, interesting to note that Irish liver swabs (Table 7) which yielded a high positive rate were taken during December, 1950, and January, 1951. This discrepancy is being further investigated.

Samples of *bile*, 116 Irish and 218 non-Irish, were examined, and of these, four Irish and one non-Irish yielded *Salm. dublin*, percentages of 3·4 and 0·5 respectively (Table 1). As the proportion of positive isolations (Irish to non-Irish) was similar to that obtained in the examination of faeces, the investigation of bile samples was discontinued.

Liver swabs, on the other hand, gave very interesting findings. A series of 202 Irish livers were cultured individually in a manner similar to the faeces and the results are collected in Table 7.

TABLE 7

Incidence of *Salm. dublin* in Irish liver swabs

	Dublin	Limerick	Belfast	Total
Number of liver swabs	157	29	16	202
Number positive	18	5	2	25
Percentage	11·5	17·2	12·5	12·4
Number of batches	9	1	2	12
Number of positive	7	1	2	10

It is rather surprising that out of 12 batches of Irish liver swabs only two failed to produce *Salm. dublin.* In this connection it is of interest to recall that until fairly recently the consumption of raw liver was part of the usual treatment of pernicious anaemia.

By comparison, a series of 171 non-Irish liver swabs were examined and the results appear in Table 8. There were no positives.

TABLE 8

Incidence of *Salm. dublin* in non-Irish liver swabs

	Cheshire	Denbigh	Flint	Total
Number of liver swabs	104	12	55	171
Number positive	0	0	0	0
Percentage	0·0	0·0	0·0	0·0
Number of batches	5	1	3	9
Number positive	0	0	0	0

Milk. In July, 1950, *Salm. dublin* was isolated from the faeces of three guinea-pigs found dead after inoculation with samples of milk for tubercle bacilli. A fortnight later, a fourth guinea-pig yielded a culture of *Salm. dublin.* Routine tests of guinea-pig faeces were carried out over a period of some months, but no more positives were found.

From August, 1950, until the end of the year, 762 milk deposits were cultured for *Salm. dublin*, and one positive was found. This was from a pooled deposit, and the source could not be traced; but it is interesting to note that of the milks concerned some came from the Shropshire-Cheshire-Staffordshire border where infections with *Salm. dublin* have been described in man.

Summary

Samples of faeces and bile and liver swabs from healthy cattle of Irish and non-Irish origin were examined for organisms of the *Salmonella* group. Except for *Salm. typhi-murium* on one occasion, *Salm. dublin* was the only type isolated.

From 1st November, 1949, until 31st December, 1950, *Salm. dublin* was isolated from 10 per cent. of samples of faeces and 3·4 per cent. of samples of bile from the Irish cattle examined; the corresponding figures for non-Irish cattle were 3·7 per cent. and 0·5 per cent.

Of 202 liver swabs from Irish cattle examined, 25 yielded *Salm. dublin;* of 171 non-Irish liver swabs examined, none was positive.

One deposit from a home-produced milk out of 762 milk samples examined yielded a culture of *Salm. dublin.*

Young Irish cattle on the whole showed a fairly steady infection rate, but in older animals culled from home dairy herds there was a tendency for most batches of animals to be completely clear, and for an occasional batch to give an abnormally high rate. It is hoped to follow up the reasons for this irregularity during the coming year.

We should like to thank Dr. Joan Taylor, of the Salmonella Reference Laboratory, Colindale, for help during the investigation.

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health,
23 Savile Row (Room 207),
London, W.1.
Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The October Bulletin was issued on 17th October.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

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DECEMBER **1951** **Vol. 10**

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SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1.

WINTER EPIDEMICS: A REQUEST FOR EARLY INFORMATION

This memorandum discusses the methods by which the medical officer of health may learn more promptly about a sudden rise in the incidence of influenza and similar diseases in his district, and why it would now be profitable to study winter epidemics more intensively. The need for detailed information about the epidemiological pattern seen in the smaller geographical units is obvious and it is hoped that many district medical officers of health will provide the information now sought.

*Ascertainment**

The laboratory diagnosis of virus influenza itself is now a fairly simple procedure, but clinically the differentiation of the several types of acute respiratory infections is difficult during the early stages. Even when patients are admitted to special research wards and complicated laboratory investigations carried out the diagnosis may remain in doubt. Compulsory notification of a specific respiratory disease which presents no easily recognised characteristic signs is unlikely to be of value. However, it is felt that the assembly and study of all readily available information can hardly fail to help in overcoming some of the difficulties of determining the source and nature of an impending epidemic.

Influenza is, of course, the most serious of the winter epidemics. Outbreaks may sometimes have an insidious onset: when this happens the cases are infrequent initially though usually occurring in groups. Thereafter the build-up is a rapid mathematical progression, but the incidence in a large population is seldom uniform.

The family doctor is generally the first to recognise infected groups, by a sudden increase in the number of attendances and visits. Within a few days he may find his daily list trebled or quadrupled, but it may still be several days before any death occurs or the registrars note any change. The rise in incidence may not affect all parts of a town or be recognised by all practitioners simultaneously, nor does the number of sick calls represent the total incidence. During most winter epidemics many of the patients only suffer trivial illnesses, such as feverish colds, and do not consult their doctors. They may, however, stay away from work for a day or two. Absenteeism in an industry or sickness in a residential institution under continuous medical supervision may therefore be the first tangible signs. Medical officers to residential schools are in a particularly favourable position to make observations.

Somewhat later the new claims on the Ministry of National Insurance provide the first measurable information and still later, notifications of pneumonia or certifications of death begin to rise.

The sources of information are therefore :—general practitioners ; industrial and residential communities, including units of the armed services and hospitals ; claims to sickness benefit and death grants at Ministry of National Insurance offices ; registrars of deaths ; and, in some districts, information regarding ambulance calls and demands on the Emergency Bed Service.

(Footnote: The 1951 epidemic is discussed in Proc. R.S.M., 1951, **44**, 789–802. Some of the statements made in this memorandum are elaborated in those papers. A paper by Isaacs, A. and Andrewes, C. H., (1951), B.M.J., ii, 921, is also topical.)

* Some advice on this subject was included in a note dispatched to all M.Os.H. by P.M.Os Ministry of Health on 22nd January, 1951, headed " Influenza 1951 ".

As regards practitioners, it will be recognised that the clinical appraisement of the epidemic must rest mainly with general practitioners and the medical staffs of hospitals. During the 1951 epidemic many selected practitioners provided regional medical officers with weekly reports which were used in compiling the weekly "Epidemiological Comment" published in the national medical journals.

Although this method of collecting information proved to be satisfactory, it is obviously more appropriate that it should be done through medical officers of health who will wish to keep themselves informed both of the extent and clinical features of the epidemic in their particular district. It is therefore suggested that medical officers of health should make some prior arrangements with practitioners who would act as "spotters" and furnish regular reports when the occasion arises. Medical officers of health will, no doubt, already know the names of the doctors who have shown a special interest in the problem but, if not, should consult the regional medical officer.

As regards industrial and residential communities, the medical officer of health can make direct contact with the medical officers concerned and ask them to telephone him when there is a significant increase in absenteeism from upper respiratory disease in the industry or community.

As regards sickness benefit claims and death grants, there is a standing arrangement whereby medical officers of health of counties and county boroughs may obtain from the regional controllers, Ministry of National Insurance, the weekly figures relating to new sickness benefit claims put in at the Ministry's local offices in their areas. District medical officers should arrange to get the appropriate weekly figures from the county medical officer. The Ministry of National Insurance is considering the matter further and it may be possible for district medical officers of health to receive direct from National Insurance local offices in their districts an immediate intimation when local sickness benefit claims first show a sharp rise. When this arrangement can be made, confirmation will be given later, but the information would be in any case related only to initial rises in claims. The later weekly figures will be available through the county medical officer under existing arrangements.

With regard to registrars, the Registrar General has agreed that medical officers of health may ask registrars to provide daily reports on total deaths and deaths from pneumonia, bronchitis and influenza during the emergency period. Information regarding the age at death will also be available.

The differential diagnosis of epidemic influenza from other upper respiratory infections is largely a laboratory procedure. It is therefore requested that the medical officer of health should now discuss with the directors of public health laboratories or, in some instances, hospital laboratories, the mechanism for bringing to the notice of the laboratory the very first indications of an outbreak and for providing the laboratory with the special material required. For this the medical officer of health may wish to bring the laboratory into direct contact with the general practitioners acting as "spotters" so that the necessary collecting outfits may be provided before the epidemic begins. The public health laboratories have already received a circular on this subject.

The laboratory studies will be made on selected material from the district. The results will not as a rule be available in time to be of any value to the clinician in relation to the individual patient.

When the medical officer of health decides that an unusual prevalence is to be expected, a note giving the information available should be sent to S.M.O., Med. 3, Ministry of Health, Savile Row, London, W.1.

This preliminary notice, describing the outbreak in general terms, will be of great value to the Ministry and it is hoped that most medical officers will follow up by sending reports for weeks ending on Saturdays, should the outbreak be substantial.

In the case of ports and airports, port medical officers are particularly requested to report immediately to S.M.O., Med. 3, Ministry of Health, the occurrence of influenza-like disease aboard ship or amongst passengers or aircrew arriving in this country. It is hoped that similar reports will be made by the armed service medical authorities concerned in the case of aerodromes and ports under service control. Arrangements for investigation will be made according to the circumstances.

Reasons why a more intensive study of winter epidemics is opportune

1. Immunisation would appear to be the most hopeful way of preventing and combating influenza. During the past few months two important observations have been made.

(*a*) The addition of oily adjuvants to egg vaccine enhances antibody production phenomenally.

(*b*) More is now known as to which antigenic strains of influenza virus are likely to be best for incorporation in vaccines.

The Ministry of Health has decided to support the production of influenza virus egg vaccine and the Medical Research Council will soon announce the appointment of a committee to study the technique of vaccine production and administration and to promote extensive field trials. The present difficulties of immunisation against influenza mainly arise because of our ignorance of certain of its epidemiological features. Further study of these problems is necessary before virus vaccine can be used with success on a large scale.

2. The establishment of the World Influenza Centre at the National Institute of Medical Research, Mill Hill, and of numerous other national centres throughout the world has, during the past three years, brought a great volume of new knowledge about the global epidemiology of influenza suggesting that the movement of the disease across the world can now be traced and forecasts made. Opportunities to study in greater detail the spread through this country are now sought.

For example, although Newcastle suffered a particularly high attack rate and heavy death rate early in 1951 and there are reasons for believing that the disease came there from Scandinavia, the causative virus was not isolated. At a later date two types of virus A were recovered in other parts of the country, the disease at Liverpool being due to a strain which had characteristic differences from strains found in more easterly areas of England. The Liverpool strain may well have come from South Africa ; it is unfortunate that we shall never know whence the Newcastle strain came or whither it moved.

It is hoped that representative strains of virus will be isolated from all the main centres of population at the time of the *first appearance* of influenza. Once it has spread wide the matter is of less moment.

3. The strains of virus recovered in a given year may differ widely in antigenic properties from the strains recovered in past years. For example, strains isolated before 1947 have no immunising effect against strains recovered since. Strains recently recovered have been fairly closely related but another major change may occur at any time. To obtain an effective vaccine it will often be necessary to use recently isolated strains of virus and to administer the vaccine as soon as it can be prepared. It is clearly

desirable to identify all the epidemic strains of virus as soon as the disease appears and as far ahead of the zenith of the epidemic as possible. For this a nation-wide net is necessary because none can forecast where the first outbreaks will appear. Nor can the time of first appearance be guessed. Localised outbreaks in May and June have on several occasions been forerunners of bigger outbreaks in the autumn and winter. Sporadic cases during the summer are of special interest.

4. Recent developments in hospital services and the possibilities of improved therapy may mean that the mortality from the more severe respiratory diseases could be reduced if selected cases were hospitalised more promptly and more often. In order that the hospital boards and governing bodies can free beds for this purpose early notice of an impending outbreak must be given them. The medical officer of health will wish to provide the boards with reliable forecasts. The Ministry's medical officers will also assist in this matter.

5. With recent developments in hospital and laboratory facilities it may be possible to provide more satisfactory methods for differentiating the several forms of winter respiratory disease occurring in epidemics. Until progress is made along these lines the scientific application of specific therapy may be impossible.

Information Service and Public Relations

The public has become more insistent in the matter of information when epidemics occur, and it seems proper that the medical officer of health should deal with public relations in his own district. To assist him the Ministry of Health will try to summarise each week any information available and publish notes in the weekly medical journals when there is a high prevalence.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

NOVEMBER, 1951

(Issued from the General Register Office, Somerset House, W.C.2)

	November 3	November 10	November 17	November 24	Average weekly figures for November, 1950
Scarlet Fever	1,068	1,232	1,473	1,477	1,297
Whooping Cough	1,477	1,625	1,833	1,892	3,827
Diphtheria	46	42	38	29	60
Measles, excluding Rubella ...	2,006	1,935	2,084	2,048	9,544
Acute Pneumonia	510	479	464	430	497
Meningococcal Infection ...	33	37	42	46	32
Acute Poliomyelitis (Paralytic)...	62	56	45	41	133
,, ,, (Non-paralytic)	20	22	20	16	45
Ophthalmia Neonatorum ...	57	32	45	32	36
Puerperal Pyrexia and Puerperal Sepsis	242	220	238	243	64
Dysentery	175	192	240	221	443
Paratyphoid	26	10	14	14	4
Typhoid	2	4	4	5	3
Smallpox	—	—	—	—	—

No cases of Cholera or Plague.
One case of Typhus Fever (not louse borne) imported. In week ended 24th November.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street,
Westminster, S.W.1.

A DOUBLE-INOCULATION SCREENING TEST FOR TUBERCLE BACILLI IN MILK

R. D. Gray, M.D., D.P.H., J. H. Evans, F.I.M.L.T., and
Gwynneth V. Pearce, A.I.M.L.T.

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Few public health laboratories in this country have sufficient animal house accommodation to enable them to make frequent and regular biological examinations of all the milk sold without heat treatment in their area. The general practice is to restrict sampling for this purpose to selected groups, such as Accredited supplies, raw milk supplied to schools and institutions, or milk produced in an area where the incidence of infected herds is unduly high. By so limiting the number of tests one can conduct each examination by the orthodox method (Wilson and Miles, 1946) whereby two guinea-pigs are devoted to each sample, and in Cambridgeshire it has been possible to ensure that bulk samples are collected from groups of not more than ten cows (McCoy, 1950). In this way a proportion of the untreated supplies can be examined for tuberculous infection with the animal test working at its greatest efficiency.

The problem has for long been tackled differently in Newport. At the request of the Monmouthshire and other health authorities, sampling has always been extended as widely as possible, and, since the provision of the animal house in 1920, successive laboratory directors have aimed at examining biologically every sample of raw milk received. The inevitable compromise had, therefore, to be made in the laboratory, and from the outset only one animal was used for each routine test. As the scheme expanded, and partly for financial reasons (when local authorities paid *pro rata* for biological examinations), a stage was reached when a composite sample of the bulked milk from 2-3 farms was inoculated into one animal. To this practice there were the following objections: (1) the use of only one animal increased the risk of failure through premature death of the animal, (2) a negative result was the less trustworthy because of the increased dilution factor, and (3) a positive result was somewhat embarrassing to the veterinary inspectors, since two or three farms, rather than one, had to be searched for the infected cow.

Principally to overcome this last objection, the suggestion was made in 1936 by one of us (J. H. E.) to adopt a double-inoculation technique whereby the sample from one herd was inoculated into the right groin, and the sample from another herd inoculated into the left groin, of the same guinea-pig. As a preliminary trial of this technique proved satisfactory, the method was adopted as the routine screening test and has now been in continuous use for fifteen years. Although entailing the same risk of premature death, this method was expected to give more reliable negative results in that, the dilution factor being lower, there should be a greater chance of detecting an infected animal. Any advantage in this respect would be far reaching, as most of the results were " double-negatives," i.e., negative on both sides. When a " positive-negative " result occurred, i.e., positive on one side and negative on the other, the Animal Health Division of the Ministry of Agriculture, besides following up the positive result as usual, took from the " negative " farm a bulk sample which was biologically tested by the orthodox two-animal method. This latter practice was discontinued after a period of years when it was realized that every negative screening test had been followed by a negative orthodox test. In the very few instances where a " double-positive " result has occurred, the subsequent clinical investigations have usually produced evidence of infection in both farms.

The Double-Inoculation Technique

The inoculum originally used was 1 ml. of the centrifuged deposit from 100 ml. of the sample. Since 1948 the gravity cream has been incorporated, the inoculum becoming 1·5 ml. of a mixture of the gravity cream and the centrifuged deposit. The inoculum from one sample is injected subcutaneously into the right groin of the guinea-pig over the inguinal glands, care being taken not to pierce the peritoneum. Long experience has shown that this method only exceptionally leads to skin ulceration. The inoculum from another sample is similary injected into the left groin of the same animal. This procedure is used for all routine samples of raw milk arriving in the laboratory, so long as an even number of samples is received. Not infrequently, of course, there is an odd sample which has to be inoculated alone into one animal.

Samples collected by the Animal Health Division and submitted specifically for a biological test are generally given the orthodox two-animal test. Occasionally, when animals are scarce, only one animal is used for each test.

Animals surviving six weeks from the time of inoculation are killed and examined for signs of tuberculosis. Those dying prematurely are also examined in case signs of tuberculosis have developed. Negative results after less than three weeks' survival are rejected as an incomplete test. Negative results after death at 3-4 weeks are accepted with caution.

Positive signs of tuberculosis usually develop from 3-4 weeks onwards and are generally well advanced by six weeks. These consist of enlargement and caseation of the inguinal glands on the affected side, enlargement followed by caseation of a sublumbar gland, with the appearance of tuberculous foci in the spleen and later in the liver. All lesions are confirmed by microscopic examination and a positive diagnosis is not made unless caseation is present in at least one gland and unless acid-fast bacilli are found in the caseous material.

In the double-inoculation method a positive sample is distinguished from a negative sample only by the location of the affected inguinal glands, those on the one side being enlarged and caseous while those on the other side are normal. Fortunately, evidence of tuberculosis has with one exception always been accompanied by involvement of the inguinal glands. In this exception it appeared probable that the peritoneum had been punctured during inoculation.

RESULTS

Comparison of the orthodox and double-inoculation methods

From the foregoing it will be evident that for the past fifteen years the following four procedures have been in use:—

1. Animal Health Division (A.H.D.) samples: one sample into two animals.
2. Animal Health Division (A.H.D.) samples: one sample into one animal.
3. Routine samples: two samples into one animal.
4. Routine samples: one sample into one animal.

Records prior to 1944 are regrettably no longer available, but it was felt that those for the past seven years might provide sufficient evidence for an assessment of the screening test. The validity of a positive and of a negative screening test had already appeared satisfactory both to us and to the Animal Health Division veterinarians, but what still required long-term comparison was the proportion of failures arising from premature death of the animal. The results are shown in Tables 1 and 2.

TABLE 1

Animal Health Division Samples

Year	One sample inoculated into two animals					One sample inoculated into one animal				
	Total number of samples	Positive results (one animal or both showing evidence of tuberculosis)		Failures (both animals dying in 21 days or less without showing evidence of tuberculosis)		Total number of samples	Positive results		Failures (animal dying in 21 days or less without showing evidence of tuberculosis)	
		No.	Percentage	No.	Percentage		No.	Percentage	No.	Percentage
1944 ...	218	16	7·3	12	5·5	0	—		—	
1945 ...	162	7	4·3	0	0·0	0	—		—	
1946 ...	177	16	9·0	2	1·1	13	1		0	
1947 ...	81	8	10·0	2	2·5	53	4		4	
1948 ...	142	11	7·8	5	3·5	54	3		5	
1949 ...	193	13	6·7	2	1·0	0	—		—	
1950 ...	146	8	5·5	5	3·4	15	0		4	
Total...	1,119	79	7·1	28	2·5	135	8	5·9	13	9·6

By both procedures:

Total number of samples	1,254
Total positive results	85; percentage 6·8
Total failures	41; percentage 3·3

TABLE 2

Routine Samples

Year	Two samples inoculated into one animal					One sample inoculated into one animal				
	Total number of samples	Positive results		Failures (animal dying in 21 days or less without showing evidence of tuberculosis)		Total number of samples	Positive results		Failures (animal dying in 21 days or less without showing evidence of tuberculosis)	
		No.	Percentage	No.	Percentage		No.	Percentage	No.	Percentage
1944 ...	678	4	0·6	120	17·7	68	1		18	
1945 ...	530	4	0·8	60	11·3	36	0		5	
1946 ...	446	5	1·1	66	14·8	35	0		4	
1947 ...	268	2	0·7	26	9·7	40	1		2	
1948 ...	366	2	0·5	36	9·8	61	2		10	
1949 ...	724	18	2·5	86	11·8	129	7		6	
1950 ...	622	6	1·0	90	14·4	75	2		12	
Total...	3,634	41	1·1	484	13·3	444	13	2·9	57	12·9

By both procedures:

Total number of samples	4,078
Total positive results	54; percentage 1·3
Total failures	541; percentage 13·3

It will be seen that the A.H.D. samples show a much higher percentage of positive results (average: 6·8 per cent.) than the routine samples (average of positive results: 1·3 per cent.). This should not be attributed to the greater delicacy of the orthodox test since the samples in Table 1 are not, from this aspect, comparable with the samples in Table 2. Whereas the routine samples are representative of all the milk sold without heat-treatment in and around Monmouthshire and are submitted without any suggestion of tuberculous infection, the A.H.D. samples are submitted chiefly because of clinical or other suspicion of tuberculosis. The percentage would doubtless be higher still but for the fact that the Animal Health Division examinations include a large number of clearance tests for absence of infection after removal of the offending animal.

The superiority of the orthodox two-animal test is, on the other hand, clearly shown by the low proportion of failures—2·5 per cent.—as compared with 9·6 per cent. when only one animal was used for A.H.D. samples, and 13·3 per cent. for routine samples. Given sufficient accommodation and animals there is no doubt that universal application of the two-animal test would provide the most satisfactory results. The benefit of the second animal is further illustrated by Table 3 which shows that, of the 2,238 animals used for the 1,119 two-animal tests, 239 died in 21 days or less and yet this 10·7 per cent. premature mortality caused only 28 (2·5 per cent.) failures.

TABLE 3

Comparison of Premature Mortality with Test-Failure Rate in Examinations by the Orthodox Two-Animal Method

Year	Tests	No. of animals used	No. of animals dying in 21 days or less	Test failures
1944	218	436	77	12
1945	162	324	31	0
1946	177	354	32	2
1947	81	162	9	2
1948	142	284	30	5
1949	193	386	29	2
1950	146	292	31	5
Total ...	1,119	2,238	239	28

When only one animal is used for the test, the proportion of failures is directly dependent upon the premature mortality. Over the seven-year period under review, the total number of animals used for the biological testing of milk was 4,634(¹) and the total number dying in 21 days or less was 551(²). The total premature mortality was therefore 11·9 per cent. As this figure is close to that of the failure rate (13·3 per cent.) in the double-inoculation series (see Table 2), it would appear that the giving of two injections does not cause any material increase in the premature mortality. The failure rates in the single-animal series (12·9 per cent. and 9·6 per cent.), although the figures are rather small, tend to confirm this view. Of more consequence, perhaps, is our experience over the years that death does not occur with increased frequency during the first few days after inoculation. Most of the deaths are due to pneumonia and occur at any time during the six-week period when a sudden change in the weather unduly lowers the temperature of the animal house. As the heating of the animal house is not thermostatically controlled our best efforts to maintain an equable temperature are sometimes defeated. At such times the singly inoculated animals are no less susceptible than the others. When better accommodation becomes available it should be possible, in the absence of epidemic disease, to lower the mortality rate and so decrease the proportion of test failures.

The figures quoted by Stringer (1951) are of interest in this connection. Of 2,067 biological tests conducted at various laboratories during 1947-50, 214 (10·4 per cent.) tests were incomplete "owing to premature deaths of guinea-pigs, souring samples, etc." It would, therefore, appear that other laboratories experience a failure rate of some 10 per cent. Unfortunately the technical procedure is not described but, if our figures are any guide, it would seem that single animals rather than pairs were used in these tests.

Discussion

Though the superiority of the two-animal test is not in question, shortage of animal house accommodation, and more recently of animals, prevents this test being used for all samples. It becomes, therefore, a matter for individual discretion whether it is better to restrict the number of samples for biological examination or to adopt a less elaborate test which permits

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(¹) 2 × 1,119 + 135 (Table 1) + ½ × 3,634 + 444 (Table 2).

(²) 13 (Table 1) + ½ × 484 + 57 (Table 2) + 239 (Table 3).

wider sampling. The compromise of using a single animal for each test, though allowing double the amount of sampling, may incur a fairly substantial proportion of failures. In an effort to increase the amount of sampling still more we have endeavoured to use only half an animal for each test and our failure rate appears to be little greater than if we had used a whole animal. Moreover, as a result of the liberal sampling which this method permits, it has been possible during most of the time under review to make repeated examinations at regular intervals. The adverse effect of the inevitable failures is thus to some extent compensated.

Results from completed tests are usually as clear-cut by the double-inoculation technique as by the other methods. " Double-negative " findings require little comment. They comprise in our area at least 98.5 per cent. of completed tests and represent a very substantial saving of animals. The usual single-positive (" positive-negative ") result leaves no doubt in the mind of the examiner, there being obvious lesions in one inguinal region and a complete absence of lesions in the other.

The apparently " double-positive " results, however, require careful assessment, since infection, once it has reached an advanced stage, may spread to the opposite side. Such results are very uncommon. We have had three in the series under review. In one case where the lesions suggested a true " double-positive " result the presence of infection in both farms was subsequently confirmed. In another similar case there was no evidence but considerable suspicion, since animals had been sold from both farms before the Animal Health Division inspection. In the third case where advanced inguinal lesions were present on one side with an early inguinal lesion opposite, infection was confirmed at the one farm but not at the other.

Spread of infection to the side opposite that of inoculation is known to occur occasionally in animals receiving only one injection. McCoy (1951) found bilateral glandular lesions following unilateral inoculation twice in 118 positive tests (total tests: 4,274). We have noticed this rather more frequently in our singly inoculated animals, perhaps because we do not kill until 6 weeks after inoculation. It would possibly be wiser to palpate from 4 weeks and to kill as soon as an inguinal lesion becomes manifest.

In our experience of the double-inoculation technique, advanced inguinal lesions on both sides without liver involvement denote a true " double-positive " result. In the presence of liver involvement a small inguinal lesion opposite an advanced lesion probably represents spread rather than a second infection. Such occurrences are so few, however, that the obvious policy is to report any such result as doubtful and have it investigated further.

Conclusions

With careful record keeping and good animal care the double-inoculation method permits adequate sampling and provides results which rival the single-animal method and are inferior to the two-animal test only in the greater proportion of failures resulting from premature death of the animal.

The expansion of pasteurization is gradually reducing the number of supplies that require repeated biological testing, but the rising cost of guinea-pigs is unfavourable to the regular employment of the orthodox two-animal test. Possibly the best compromise of all would be to apply the double-inoculation technique to pairs of animals. While requiring, for the same degree of sampling, no more animals than the single-animal method, this procedure should give the added protection of the orthodox technique from test failures. We are giving this procedure a trial but have not yet sufficient animals to use it as a routine. The results are encouraging.

Summary

A double-inoculation technique for tubercle bacilli is described whereby two samples of milk can be biologically tested in the same guinea-pig. The method consists in bilateral subcutaneous injections in the inguinal region, and interpretation depends upon which, if either, set of inguinal glands develops tuberculous lesions 6 weeks later. The test-failure rate (through premature death) is about the same as when single animals are used (13 per cent.), but considerably more than when pairs of animals are used (2.5 per cent.). This defect could possibly be remedied by applying the double-inoculation technique to pairs of animals.

We wish to pay tribute to our animal attendant, Mr. T. Jennings, for his excellent services under difficult conditions during the past 6 years. We are also greatly indebted to Mr. R. C. Locke, M.R.C.V.S., D.V.S.M, Divisional Veterinary Officer, Animal Health Division, Monmouthshire, for supplying us with detailed records of the clinical investigations and for much helpful collaboration.

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